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The Beattie-Smith Lectures.[1]

(The University of Melbourne.)

LECTURE II:[2] THE PROBLEM OF SCHIZOPHRENIA.

By JOHN F. CADE,

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PERHAPS the best introduction to this lecture would be to quote to you from the poems of Eithne Tabor, a young American girl who developed schizophrenia. In a sense it is fortunate that amongst the victims of this illness are sometimes people of great artistic ability who are able occasionally by their skill of pen or brush to portray for less imaginative mortals the disordered fabric of their minds. This girl has described with extraordinary clarity various stages in the development of her disorder. Her book is called "The Cliff's Edge: Songs of a Psychotic", and the first poem quoted is called "Warning":

O'er the cliff's sheer edge
I am reeling, plunging, falling,
And out on the seas beyond
A vanished voice is calling,
For the sound of the past in my deafened ears
Is a lone lost seagull's crying;
While my manacled soul, which the gods struck down
Like Prometheus of old, lies dying. . . .

Another, entitled "Paranoid", gives vivid insight into the mind so affected:

Legions against him
None to aid,
Somewhere there's plotting,
A trust betrayed,
Outrageous fortune
Turns her sling
Against his person,
And he the King;
Ah, in his throne room
Hangs so drear
Delusions of splendour
Hiding—cold fear.

By schizophrenia I mean a syndrome characterized by five major symptoms or signs—namely, apathy, intellectual confusion, erratic behaviour, delusions and hallucinations. Apathy may be present alone as in the simple type of *dementia præcox*. This type of patient is the person who shows a steadily diminishing purposive drive, who for a while drifts aimlessly from job to job and then into chronic shiftlessness; who is for a time upbraided for laziness and exhorted to "pull himself together", until finally the realization dawns that he is not lazy but ill. On the other hand, delusions may be a solitary symptom, as in paranoia; but in the generality of patients all five are present with varying degrees of emphasis.

Although the subdivision of schizophrenia into the subgroups of *dementia præcox*, paraphrenia and paranoia is useful for descriptive purposes, it is extremely doubtful whether it has any validity. There is every gradation between simple primary dementia at one end of the scale and paranoia at the other. Moreover, the same patient may exhibit a different emphasis on symptoms at different times.

[1] Delivered at Melbourne on May 7 and 14, 1951.

[2] The first lecture was published in the issue of August 18, 1951, at page 213.

He will be described confidently as suffering from *dementia præcox* at the age of twenty years and from paraphrenia at the age of forty years. One psychiatrist will diagnose paranoia in a patient who would be described as a paraphrenic by another. The sub-classification of *dementia præcox* into simple, hebephrenic, paranoidal and catatonic varieties has even less validity. The one patient may easily have all four labels affixed to him at different times.

Ever since Kraepelin at the end of last century made his important differentiation between manic-depressive psychosis and *dementia præcox*, it has been evident that typical cases of each group are very dissimilar, and differ in symptoms, course and prognosis; but it soon becomes evident to anyone treating psychotic patients that a proportion of them have so-called "mixed psychosis" with symptoms of both groups. It is sometimes impossible to determine whether a restless and excited young person is suffering from true mania or catatonic excitement, or whether a depressed and delusional patient should be regarded as suffering from paraphrenia or melancholia. In addition a small percentage of patients with undoubtedly schizophrenic illness exhibit a cyclical course of regular remissions and relapses, just as manic-depressives do so much more commonly.

These cases of periodic catatonia are very important from two points of view, which I will elaborate later.

There are four useful bases of validity:

1. Ætiological foundation. This is the most fundamental, but it is obvious that a classification cannot be based upon an unknown cause, as in the present case.

2. Symptomatology. If symptoms are clear-cut and constant and adhere in an easily differentiated syndrome, it is probable that we are dealing with a single disorder or disease; but the more variable and inconstant they are the less can we be sure.

3. The course of the illness. This, if fairly definite, is useful from the point of view of both diagnosis and prognosis; but in schizophrenia it is highly variable.

4. The therapeutic test. The same remarks apply—that is, if results are good and clear-cut the test is of great value; but alas! once more the results of all treatments so far used in schizophrenia are notable mainly for their variability.

From all these observations it will be evident that the validity of classification of these major psychoses rests on a most insecure basis. It is quite possible that they are merely different reactions of various personality types to the one underlying disturbance. It is easy to see how one person will react with depression to adverse circumstances, another will sink into indifference, whilst yet another will become fearful or irrationally excited.

You will see, then, that from the very start we are handicapped heavily by not knowing the limits of our problem. Are we dealing with a nicely clear-cut disorder with a single cause, or with a syndrome analogous to the symptoms of anæmia with many possible causes, or have we merely a number of different personality reactions to a constantly present adverse factor?

The history of the search for the cause of schizophrenia mirrors very faithfully the major trends in general medicine during the last fifty years. It was natural that as research into organic causes of disease had produced such spectacular results in the nineteenth and twentieth centuries, the techniques of morbid anatomy and bacteriology could be expected to yield fruit when diligently applied to psychiatric problems. They did do so, in fact, and various organic causes of mental disorder could thus be split off from the main body of mental illness; but schizophrenia successfully defied these onslaughts and jealously guarded her secrets. Mott was probably the most prominent early protagonist of an organic pathology of schizophrenia. His findings of sex gland atrophy are still quoted, but not very confidently, since it has been shown that inanition and acute physical disease can produce similar appearances. He also described various non-specific changes in the nerve cells of the cerebral cortex and basal ganglia, such as nuclear swelling, vacuolation, disappearance of Nissl granules and increase of lipoid granules.

Alzheimer claimed to demonstrate loss of nerve cells in chronic schizophrenia, especially in the frontal cortex. There were nuclear swelling, shrinkage of the cell body, increased lipoid and generalized gliosis. Others have described this gliosis, often reported to be focal in distribution. This type of investigation continues, but with not very characteristic and not very convincing results. A recent example is that of Winkleman and Book (1949). They state that they have observed a general decrease in the number of nerve cells especially in the anterior half of the brain, and that the most constant change was so-called chronic cell disease including shrinkage and the presence of ghost cells. They hold, too, that a common feature of the schizophrenic brain is the presence of numerous small areas where neurons have either disappeared completely or are in the process of disintegration.

Other observers have failed to substantiate these findings, and Dunlap has summed up the position as follows (Henderson and Gillespie, 1946):

> Schizophrenia is lacking in any fundamental or constant alterations of nerve cells. Such alterations as are found are probably a reaction to various bodily conditions plus post-mortem and technical factors.

I believe that much published scientific work is vitiated by failure to appreciate the range of variability of the normal and by inadequate comparison therewith. I believe, too, that this applies with particular force to statements concerning the morbid histology of the brain. The cellular architecture is so varied, and the microscopic picture depends so much on the nature and duration of the terminal illness, on the age of the patient, on post-mortem changes and on methods of preparation and staining, that it appears to me as if no one should be permitted to publish a paper on cerebral histopathology until he has been constantly engaged in studying it for a minimum of ten years.

Various observers have been struck by the very small hearts and aortas of a number of schizophrenics. Undoubtedly this cardio-aortic hypoplasia is relatively common, but it is impossible to judge what its significance may be. Other schizophrenics are quite within normal limits in this respect. This is but one of a number of attempts to correlate physical or psychological variations or types with particular forms of mental disorder. One of the best known is that of Kretschmer. He held, briefly, that those of athletic, asthenic or dysplastic habitus were more inclined to introversion and schizophrenic illness, whilst the short, thick, so-called pyknic type of individual was more prone to extroversion and to manic-depressive insanity. Whether this is statistically true or not I do not know, but it matters little, for it is common to see long, thin, manic patients and short, thick-set schizophrenics. My experience is that it is impossible to correlate physical type and mental disorder—you can find any type of mental disorder in a person of any type of physique. Tallness and shortness, "thickness" and "thinness", introversion and extroversion, are all pairs of extremes. The majority of people are in between, in the same way as most people are neither very intelligent nor very dull. To try to classify people as either introverts or extroverts is like trying to classify them as either genius or imbecile, or as either tall or short.

In addition to the approaches already mentioned, there have of course been innumerable physiological and biochemical studies which, like these others, have been largely inconclusive. One of the most important biochemical investigations was the painstaking work of Gjessing upon metabolic changes in patients with periodic catatonia. He held that total nitrogen excretion was increased in the "bad" phases, whether of excitement or stupor, and that there was nitrogen retention during "good" phases. This anabolic phase continued until just before the "bad" phase commenced, when breaking down exceeded anabolism once more. But it is not so simple as all that, for Gjessing himself describes cases in which the very reverse metabolic picture occurs—that is, nitrogen retention in the "bad"

phases and excessive excretion in "good" phases—and also other cases in which there is no relationship whatever between mental state and nitrogen excretion. A favourable therapeutic action of thyroxine in periodic catatonia is held to exist by Gjessing. After depletion of the nitrogen stores of the body by thyreoid treatment there was no recurrence of the active, stuporose or excited phases, nor were there further alternating swings of positive or negative nitrogen balance.

Hardwick and Stokes (1940-1941) were able to confirm some of Gjessing's findings, but observed that the swings in nitrogen balance were related to the state of general nutrition and could be avoided by improved nutrition without interference with the rhythm of the mental illness.

At this stage it is worth recording the chequered career of thyreoid treatment in psychiatry. It was introduced by the Scottish psychiatrist Lewis Bruce, and it was for many years practically the only treatment for *dementia præcox* that appeared to be of the slightest use. Thyreoid was used in huge doses, and rather surprisingly serious toxic symptoms were rare in schizophrenics even with these big doses. In some cases it did seem to give good results; but when insulin shock treatment was introduced into this State in 1937 its much greater effectiveness quickly led to thyreoid's being completely discarded and in fact forgotten. It was Gjessing's work which later partially revived thyreoid as a therapeutic weapon. It is undoubtedly of great value in that small number of schizophrenics whose illness is truly cyclical—the periodic catatonias. It is probably the most valuable treatment and the only one that is likely to restore the patient to normal. Hence, as I said earlier, the importance of differentiating this group (a small one) from the general body of schizophrenic patients. For these latter—the great majority—thyreoid is practically useless. It may take several months of treatment with thyreoid before the normal phases have been stabilized and the "bad" phases suppressed, so it is important not to discontinue treatment before two or three months' trial. It is doubtful how long treatment should continue after the patient has become normal, but it does seem wise to continue for at least a year after the patient leaves hospital. The following is an illustrative case history:

A young man, aged twenty-five years, had a four years' history of schizophrenia of cyclical type. He had had two courses of full coma insulin therapy without notable benefit and also electroconvulsive therapy. He was by early 1950 in the "chronic" ward of a mental hospital, and there it seemed he would remain. He would be comparatively well for a week or ten days, but for the next fortnight he would be confused, hallucinated, delusional and aggressive. His "bad" phases were becoming longer and his periods of relative normality shorter. In April, 1950, he was started on thyreoid treatment (10 grains of fresh gland tablets three times a day). There was a latent period of about six weeks when little or no improvement was noted, and I was on the point of discontinuing treatment. But from that time on there was steady improvement over about two months, when he had progressed to complete normality. He left hospital early in November, 1950, and has now been working satisfactorily for some months. He continues to take thyreoid. How long he should do so, I do not know.

Another biochemical investigation of possible importance is the determination of the distribution of enzyme activity within the central nervous system. Winifred Ashby (1950) has studied the quantitative distribution of carbonic anhydrase, an enzyme which catalyses both phases of the reaction $CO_2 + H_2O \leftrightarrows H_2CO_3$ and is therefore of importance in the transport of carbon dioxide. It is a stable enzyme, readily studied in autopsy material, and there seems to be a relationship between its concentration and functional dominance. Ashby has studied its distribution in the brains of both sane and psychotic patients. Her work suggests that deviations from a normal enzyme distribution exist in mental disorder, consisting of a decrease in certain areas, others areas being left with a proportionately greater content. Peripheral nerves contain no carbonic anhydrase, whereas there is a considerable amount in the cerebral cortex. In addition there appears to be a close relation between the appearance of the enzyme in the cerebrum and the onset of function. It is interesting to note that zinc, one of the latest trace elements shown to have a definite function in the body, forms the prosthetic group of this enzyme.

In recent years interest has been displayed in the question of possible disturbance of liver function in schizophrenia. Lingjaerde (1934) reported disturbances of liver function evidenced by ketonuria, increased urobilin excretion and various positive results to turbidity tests. Quastel and Wales (1938) first reported decreased synthesis and excretion of hippuric acid by catatonic patients, but this has not been confirmed by later workers. Oltman and Friedman (1950) found that the incidence of positive results in cephalin-cholesterol flocculation tests was almost identical amongst catatonic patients newly admitted to hospital, non-catatonic schizophrenics and other psychiatric patients. Results with a chronically ill group were somewhat equivocal. The results of thymol turbidity tests for chronically ill patients were essentially negative. These workers concluded that their study lent no support whatever to the theory that liver damage is a factor in the production of catatonia or other schizophrenic symptoms. During the last twelve months or so I have studied the urinary urobilin excretion of various schizophrenics by a rough quantitative method. The amount excreted by "chronic" patients was quite within normal limits, as was also that excreted by non-schizophrenic patients. But in a small group of schizophrenics whose symptoms fluctuated either spontaneously or as a result of electroconvulsive therapy there was a similar fluctuation in urobilin excretion. When they were deteriorating, urobilin excretion was greatly increased; when they were improving, urobilin excretion was reduced, usually to within normal limits. Increased urobilin excretion, of itself, does not necessarily spell liver damage. This does seem a promising line of investigation, as in some cases at least there appears to be a correlation between mental state and urinary urobilin excretion.

Apropos this small and very preliminary investigation is the question of suitability and mass of material. It is a mistake to think that mass of material alone has any particular virtue or necessarily carries conviction. Single samples from 1000 patients may convey far less information of value than, say, 100 serial samples from 10 carefully selected patients. In other words, a cross-section may or may not reveal as much as a longitudinal section. Both should be made. The first or cross-section represents the static approach, the second or longitudinal section the dynamic. In the first case the cross-section will reveal variations of a particular phenomenon from person to person, whilst the longitudinal section will show variations in the same patient at different times and in different circumstances. If, for example, urobilin excretion is greatly increased only when a schizophrenic patient's condition is rapidly deteriorating, then investigation of both normal people and of already deteriorated schizophrenics may reveal no difference in output. It is possible, therefore, that two investigations of the same phenomenon may both give correct results within limits, and yet reach apparently contradictory conclusions. One has taken a cross-section, the other a longitudinal section.

Let us now turn to another field of medical research which may yet be applicable to our own especial problem of schizophrenia. Since Hench's work on the dramatic effect of cortisone and ACTH on rheumatoid arthritis and a host of other conditions, world-wide attention has been focused on the patient work of Professor Hans Selye of Montreal and his conception of the adaptation syndrome and stress disorders. For many years the Meyerian school has thought of schizophrenia as the end result of a long succession of frustrations and maladaptations. It would be a magnificent vindication of this theory if schizophrenia could be explained in terms of Selye's stress disorders. It would also open wide the gates for therapeutic success. Let me hasten to add that so far these hopes have not been fulfilled in the least degree. Some work has already been published and no doubt much is at present being done in an attempt to apply Selye's hypothesis to the

explanation and treatment of the major problems of schizophrenia and depression.

McNee (1950), after watching in America most of the clinical and experimental work with cortisone and ACTH, stated that "the almost immediate euphoria and mental stimulation in the patients is equally remarkable and there are now good reasons for believing that this is not just an ordinary psychological effect but if kept going by continual treatment may in rare cases end in acute psychosis". He mentions, too, that he has heard of violent mental disturbance as one of the serious side effects.

Ashby (1949) investigated the urinary excretion of cortical steroids following electroconvulsive therapy. She found that in most cases there was a brisk outpouring during the first few days and that clinical improvement was greatest in those whose steroid excretion was increased the most. Pincus and Hoagland (1950) have investigated the adrenal cortical responses of persons subjected to various stress situations. They showed that schizophrenics demonstrated striking inability to respond to test stresses with enhanced steroid output, although their resting excretion was not different from that of normal controls. Further, the locus of defective response in schizophrenics lies in the adrenal cortex, for their response to adrenal cortical extract is approximately the same as that of controls, but there is an impaired response to ACTH in the majority. Psychoneurotics, by the way, respond in normal fashion to ACTH. Unfortunately, Pincus and Hoagland did not use insulin hypoglycæmia amongst their stress-producing agents. It may have thrown light on the insulin-resistiveness of certain patients and the truly enormous doses necessary to bludgeon them into coma. Ever since insulin coma was induced for psychiatric treatment, it has been obvious that one of its effects clinically is to produce an acute outpouring of adrenaline in an attempt by the body to combat the artificial lowering of the blood sugar level; but it is now becoming increasingly probable that gluco-steroids of the adrenal cortex are also called forth in large amounts in a compensatory reaction. Is this whipping up of adrenal cortical activity the essential factor in inducing clinical improvement? The intensity of the adrenal cortical response may well determine the size of the coma dose of insulin. One might go further and suggest that cases of irreversible coma were due to failure or exhaustion of gluco-steroid response.

Already reports have been published on the effects of ACTH upon various psychoses, both depressive and schizophrenic. Not only was there no significant improvement, but some patients became worse. Cleghorn *et alii* (1950) treated eight depressives with ACTH. There was no improvement worth noting, although the activation of the adrenal cortex by ACTH was considerably in excess of that produced by electroconvulsive therapy, to which these patients later responded. It seems improbable, therefore, that electroconvulsive therapy causes its beneficial effect by increasing adrenocortical activity, as was suggested by Ashby's work just quoted.

A recent study of water and salt balance in a case of cyclical insanity showed a retention of water and salt in the depressed phase as compared with the manic. It is probable that alterations in adrenal cortical activity may have been responsible. Lehmann *et alii* (1950) also report a study on the adreno-cortical activity in a case of cyclical insanity, and found increased activity during the depressed phase and reduced activity during the manic. This "knocks on the head" the suggestion that there is a relationship between increased adreno-cortical activity and the manic state.

All these investigations point to the possibility that alterations in the activity of the adrenal cortex may play some part in the production of the final clinical picture presented by the manic-depressive and the schizophrenic; but whether such changes are primary, coincidental or secondary it is impossible, in the present state of our knowledge, to judge. The present position, then, is that we have a promising line of investigation, but it has not yet yielded results of any therapeutic value—rather the reverse; mental side-effects have been reported from the use of cortisone, both manic and depressive states having occurred, and in rare instances schizophrenia seems to have been intensified or precipitated. Premonitory symptoms have been pronounced insomnia, exaggerated mood swings and increased psychomotor activity. With regret, then, we must admit that so far the unfolding secrets of the adrenal cortex have had no immediate application in our field. I have dealt with this subject in some detail simply because it is so much to the fore both in the medical and the lay Press.

It is very much easier to appraise the past than the present, so let us go back twenty years or so; we find that the then fashionable theory of focal sepsis had its ardent protagonists in psychiatry. Schizophrenics were being cured right and left (it was claimed by some enthusiasts) by removal of infected teeth and tonsils and treatment of infected antra and sinuses. Those who obstinately persisted in remaining uncured obviously had a focus elsewhere in gall-bladder, prostate, appendix or renal pelvis, which accounted for their complete refusal to be cured. Extraordinarily impressive figures were published. However, other more critical observers were never able to duplicate these results, and for years now I have not heard of focal sepsis causing a single case of insanity, although of course we all know that an acute infection may produce toxic confusion. I have, for example, seen the unfortunate case of the certification of a person suffering from toxic confusion due to perinephric abscess.

So far we have considered various organic theories of the origin of schizophrenia, and you will agree that we have not progressed very far. At this stage we may well consider a broad classification of theories of origin. They can be placed in three broad categories and represented briefly and diagrammatically as in Figure I.

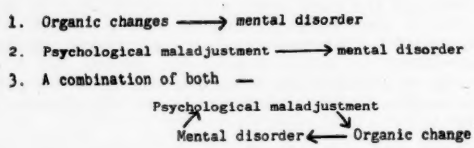
1. Organic changes ⟶ mental disorder
2. Psychological maladjustment ⟶ mental disorder
3. A combination of both —
Psychological maladjustment
Mental disorder ⟵ Organic change

FIGURE I

This third means the formation of a really vicious circle.

In addition, both hereditary and environmental influences may be operative through either of the two chief pathways. For example, Huntington's chorea has a high degree of hereditability, yet the mental disorder is undoubtedly due to organic changes in the brain. Again, it is very probable that such a purely psychological quantity as temperament is largely inherited, and this is a major factor in determining reaction to environment.

It is very easy to see how psychological maladjustment can cause minor mental maladies—the psychoneuroses—but equally difficult, for me at any rate, to see how it can cause a phenomenon such as schizophrenia. But some psychiatrists are convinced that an explanation of schizophrenia in terms of a pure psychopathology is possible, and—what is far more important—that treatment guided by such concepts will be successful. Schizophrenia, by them, is regarded as the end-result of a progressive maladaptation of the individual to the environment. Successive frustrations have led to a steady withdrawal from contact with reality and to the substitution of faulty habits of reaction. If this view is correct, that schizophrenia is essentially psychogenic, then certain conclusions follow: (i) It should be preventible by the widespread inculcation of healthy habits of mind—for example, by the great extension of child-guidance clinics. (ii) Its incidence should vary from culture to culture, according to whether or not the personal needs, aspirations and happiness of the members of a community are achieved. (iii) It should be possible to treat it by purely psychological means.

As for the first, it has yet to be proven; but I may remark in passing that the offspring of psychologists and psychiatrists have not yet achieved a reputation for outstanding stability.

The second proposition is most important. In what ways may environment determine the incidence of major psychoses, and at the feet of what particular environmental factor can the blame be laid? A partial answer to this question is given by Carothers (1947), who compared the incidence of various psychoses in Kenya natives with their incidence amongst American Negroes. The latter are approximately 50 times more liable to major psychotic illnesses. Schizophrenia is 45 times commoner among them than among the Kenya natives. Carothers concludes that the incidence of insanity amongst Africans living in their natural environment is probably very low. He considers that the clue lies in the native culture, thinking and attitude to life. To quote:

> The consciousness of being an organic and well protected member of a group gives the individual a definite self-consciousness and dignity . . . he does not suffer from social disabilities, nor is there a distinction between master and servant, rich and poor. Hence nobody suffers from an inferiority complex . . . the prevailing feeling is one of equality. . . . He feels himself part of a larger organism and enjoys observing the rules, as they give him a feeling of correctness and security, and he does not like thinking for himself. . . . A man does not plan his life, set himself an aim and exercise his strength in attaining it—personal responsibility is avoided wherever possible. In contrast to his social and sexual self-confidence, the primitive is profoundly conscious of the limits of his knowledge of nature and of his power to control his natural environment. An attitude of humbleness and awe results from this, and an implicit faith in higher powers . . . who can be approached by his social group. If this is religion, then religion is far more real to the primitive than to the modern European.

If indeed adverse cultural factors are important in the production of psychoses, and this study certainly suggests that they are, then the modern western world, with its fiercely competitive spirit, its emphasis on material values, and its absence of any real philosophy of life, must be an ideal medium for their development.

As for the third proposition, it has always been considered impossible to treat schizophrenics by purely psychological methods, apart from the occasional patient in whom psychological precipitating factors have been very obvious and could be dealt with satisfactorily. I myself have always been too pessimistic of worth-while results even to attempt anything in the way of formal psychotherapy. But now my scepticism has been dealt a heavy blow. Let me tell you about the work of Rosen—it is truly impressive. Rosen (1946, 1947) has applied certain basic psychoanalytic concepts to penetrate the psychotic systems of individuals in acute catatonic excitement. He has attempted to aid them to reestablish contact with reality through the medium of their contact with him. He deliberately assumes the identity of the figures which appear to be threatening the patient and reassures the patient that far from threatening him they will love and protect him. He believes that the excited catatonic is, as it were, in a continuous nightmare, built around an enormous fear. The patient's utterances give some clue as to what or whom he mortally fears. One must try to personify his terrifying images and convert them into protecting images. An absolute essential is the deepest possible knowledge of the unconscious. One must "converse with the patient in the language of the unconscious" and interpret it to him at every available opportunity. Each symptom, every remark, every symbol must be untwisted. Many misinterpretations may be made but these appear to do no harm—the correct interpretation strikes deep. The physician must give to the patient the same quality of love that a good parent gives to its child. He must make up for the tremendous deficit of love experienced in the patient's life. This is quite essential if the method is to succeed. Indeed, it is possible that it really is the essence of the method, and that all else—the interpretations, the psychoanalytic concepts—are unessential elaborations.

In his second paper Rosen describes the successful treatment of 37 patients with so-called deteriorated schizophrenia. This bears out the view of various eminent psychiatrists, such as Bleuler, Henderson and A. Lewis, all of whom have considered that mental deterioration is more apparent than real in *dementia præcox*. There is evidently an absence of irreversible change, and therefore always hope that a complete recovery is possible in the light of future knowledge.

Rosen admits that his results must still be regarded as tentative and that many problems have yet to be solved. Not the least important is the enormous amount of time required. Rosen, in the case of the first patient he describes, was, except for half an hour, with him continuously for sixteen hours, reassuring, feeding, protecting. Apart from therapeutic skill, such a method must demand extraordinary qualities of sheer physical endurance and courage. It requires no mean degree of equanimity and self-confidence to remain closeted alone with a wildly restless, fearful and impulsive patient for even a short time. It may be that these very exceptional qualities are the secret of success, rather than the theoretical conceptions; for to infer that these conflicts and fears are the cause of the psychotic episode is not necessarily true. It is equally possible that they may become manifest because the mind is disordered.

No physician would deny that success in treatment of many patients depends as much on qualities of heart as of mind. This is all the more true of a disorder such as schizophrenia, of which the ultimate cause is at present hidden from our view. In the assessment of therapeutic success this is a chief factor, which is not usually known to us when we peruse a paper. We may be unable to decide whether success is due to the man or to the method, but are usually inclined to give credit to the method.

The influence of heredity on the development of schizophrenia is a subject upon which many divergent opinions have been expressed and widely varying statistics published. When individual cases are considered, some occur as isolated instances in a perfectly clear family tree, whereas other families are heavily loaded with affected individuals. Neither does the family history help much in assessing progress in particular cases, for a schizophrenic with a poor family history may stage an excellent recovery, whilst another, the sole afflicted person in an apparently sound family, will steadily deteriorate.

Whatever the effect of heredity, it does not touch the heart of the problem merely to shift the blame on to one's ancestors. To say that a person is schizophrenic because one of the parents was schizophrenic is no explanation at all; it is merely a statement of fact. We are no nearer understanding the essential cause. For all that, let us examine the facts, because when relatives of a patient want to know what are their chances of developing or transmitting the illness it is important to be able to give them information about hereditary possibilities. Approximately one in 20 of the brothers and sisters of schizophrenics will develop the disorder. If one parent is schizophrenic, one in eight or nine of the offspring will be affected; but if both parents are schizophrenic just over half the children will be. This liability is considerably less than in manic-depressive insanity, and very much less than in Huntington's chorea, in which it is very high indeed. Kallman (1946) gives considerably higher figures than I have quoted. He studied the incidence of schizophrenia in similar and dissimilar twins and found that, with similar twins if one was affected so was the other in six cases out of seven, whereas with dissimilar twins the figure was one in seven—that is, about the same incidence as he found for other brothers and sisters.

You will see from all this evidence that the real cause of schizophrenia at present eludes us. Investigations have been made with every conceivable technique available to present-day medicine. The pathologist, physiologist, biochemist, psychoanalyst and eclectic psychiatrist have done their best. A number of observations have been made, and

results claimed, which have not been confirmed by further search. Results of treatment have certainly improved, but we do not seem to be any closer to knowing why, for example, one patient will stage a brilliant recovery when subjected to full coma insulin treatment whilst another remains untouched and drifts into a state of chronic psychosis despite our best endeavours.

It may be that the clue and the method lie ready to be seized at this moment, or it may be that the solution of our problem awaits advances in other fields of medicine or the development of new instruments or techniques of research. We cannot know these things except in retrospect.

What stands out from all this is the lesson I attempted to drive home in the first lecture—the prime necessity for active and widespread clinical research in psychiatry. Only thus can we hope to banish the nightmare of schizophrenia.

Now although we do not know the cause of schizophrenia we must never sink into a state of therapeutic despair or torpor. Even with our present limited knowledge much may be done. No schizophrenic should ever be labelled as incurable. I believe that all schizophrenics are potentially curable. The changes are not irreversible; no irreparable organic damage has occurred. It is well known that patients who have been sunk in a state of apparent dementia for many years have, under the influence of some stimulus, usually an acute infection, regained for short periods a state of comparative lucidity. The capacity for normal mental functioning cannot therefore have been lost beyond recall. The key is waved tantalizingly before us from time to time, and then removed swiftly before we can grasp it and use it for all. The mind in this disorder may be likened to porridge. It develops the thick, impervious scum of apparent dementia if not kept stirred by either psychological or physiological stimuli.

To illustrate what I have just said, let me quote two cases, in one of which the stimulus was physiological and in the other psychological. The excellent results are by no means usual phenomena, but they are not particularly uncommon and every psychiatrist can quote similar cases from his own experience.

A young man was admitted to my care as a deteriorating schizophrenic, as indeed he was. He had already received the usual treatment and had completely failed to respond. As I observed him over some weeks his mental state slowly became worse. In an attempt to prevent his downward course I gave him some electroconvulsive therapy. The only effect was to hasten his progress into the depths of apparent dementia. He became mute and inaccessible, oblivious to his surroundings and dirty in his habits. This went on for several months whilst I watched in futile desperation. All attempts to arouse his interests failed dismally. Finally I decided to give him a further course of electroconvulsive therapy, as it could certainly not make him any worse then. To the astonishment of everyone, myself most of all, he rapidly staged a complete recovery and has now been quite well for two years.

The other patient was under the care of my father in recent years, and was suffering from chronic delusional insanity of a number of years' duration. His chief waking preoccupation was with the mythical murder of which he imagined he stood accused. One day my father persuaded him to occupy himself with a small job. Soon afterwards he asked to be allowed to do some casual work for a local resident. From that time his delusional ideas steadily receded into oblivion and it was not very long before he was able to leave hospital and take his place in the community once more.

Adverse comments are made from time to time about the work that patients do in mental hospitals. "Slave labour" is one of the terms of opprobrium. It should be realized that it is often a therapeutic victory to get a patient started on some useful work in ward, grounds or occupational therapy department. There is nothing more demoralizing than the "chronic" airing court. Often the persuading of a patient to take up some little occupation or duty is the first step along the highroad to recovery of mental health. Even if there is no fundamental improvement in the mental health there is certainly a great improvement in self-respect and contentment and in the morale of the hospital as a whole. Nurses and attendants are well aware of this. The task may be only a simple repetitive one like polishing a floor or washing dishes, it may be physical work in the garden or a clerical job like helping in the library, or it may be the more formal approach provided by the occupational therapy department with its many interesting crafts and deliberately planned social activities under skilled supervision.

One of the outstanding features of schizophrenia is the desocialization that occurs, and one of the great problems for the recovering patient, especially for one who has been ill for a long time, is his social rehabilitation. His illness has in many instances sapped his self-confidence and left him with a deep-rooted feeling of insecurity and inferiority. This is often accentuated by the attitude of friends, relatives and acquaintances. His relatives may adopt attitudes of over-protection and watchfulness in their fear that he may relapse. The patient sees this and it adds to his uneasiness. His acquaintances, he knows or feels, talk about his recent mental breakdown. There is a caution and reserve in their attitude towards him and this adds further to his feelings of insecurity. The effect of these attitudes upon the patient may be very harmful indeed and may make him reluctant to face the world once more. Sometimes he would sooner retire again to the security of the hospital, where he realizes that he is known, understood and accepted better than anywhere else.

It is in combating this very difficult situation that a definite programme of social activity is so necessary; first within the hospital itself, and usually under the guidance of the occupational therapy department, and secondly beyond the hospital, through the skilled supervision and advice of psychiatric social workers and of educational and rehabilitation officers.

I should like to see the same range of services available for civil patients as are available for ex-servicemen.

The patient must be given assistance all along the road to recovery, so that he can with the least difficulty resume old social roles or develop new ones.

SUMMARY AND CONCLUSIONS.

1. The cause of schizophrenia is the biggest unsolved problem in psychiatry.

2. When the cause is known and treatment fully effective we can expect to empty half the mental hospital beds of the world.

3. This emphasizes the prime necessity of constant clinical research.

4. Even with present methods the results of treatment are often very good in early cases.

5. Even in long-standing cases therapeutic perseverance is sometimes rewarded with astonishing results.

6. No schizophrenic should ever be labelled as incurable. No irreversible change has occurred. All schizophrenics are potentially curable.

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AUSTRALIAN LIFE TABLES, FROM A MEDICAL POINT OF VIEW.

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THE life tables based on the Australian mortality experience for the years 1946 to 1948 have recently been published by the Commonwealth Actuary (Balmford, 1950). Since the life table is a traditional method of describing mortality, it seems worth while to discuss Australian life tables as part of the general series on Australian mortality. It is proposed here to describe the general features of the life table, but not to enter into the more actuarial aspects of construction, such as graduation, nor to attempt a detailed analysis of the changes in mortality as revealed by the successive life tables.

The Aim of a Life Table.

The primary aim of a life table is to describe the way in which a group (or cohort) of animals would die or survive under given hygienic or environmental conditions. With animals of short life span, such as flies or mice, it is possible to note the mortality experience of a cohort throughout the whole of the life span, for a human observer may reasonably expect to survive until the last of his experimental animals has died out. Thus A. B. Hill (cited by Greenwood, 1928), in the course of some nutritional studies beginning with 269 mice, was able to note the number of survivors after various intervals of time. This type of result is the "order of dying out" of German authors. Raymond Pearl (1940) has given many other examples, chiefly the mortality experience of *Drosophila melanogaster* submitted to various laboratory conditions. In Figure I are plotted Hill's survivorship figures, as cited by Greenwood, the number of survivors from 269 mice being given by length of cage life. The observed number of survivors has been plotted in each case as a circle. Greenwood graduated the data by means of a well-known actuarial formula, that of Makeham modified after Gompertz, and his graduation is displayed as the smooth curve. The observed and graduated results are in close accord. There are several reasons why this method of obtaining an order of dying out, although very important theoretically, is unsuitable for studies on the human species. The reasons are as follows.

1. There are great administrative and technical difficulties in following a number of persons throughout their life, such as expense, migration, intervening wars, resistance of persons to any form of continued observation, and lack of continuity of interest in a project of long duration.

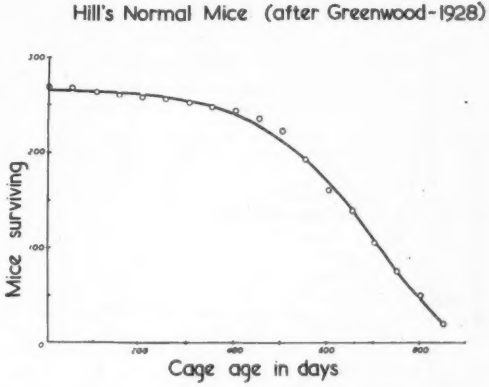

FIGURE I.

An order of dying out. A survivorship graph from data of A. B. Hill, cited by Greenwood (1928); normal mice introduced into the cage. (Ages were calculated from the time of entry of the mice into the cage.)

2. The order of dying out would not be available for some hundred years from the commencement of the observations.

3. When obtained, the mortality, represented by the order of dying out, would be that of a variable number of years prior to the time of the final entries, and so would not describe recent mortality experience, nor would it represent the mortality experience of any epoch. It would, however, represent the mortality experience of a "generation" or "cohort".

In order to obtain a summary of mortality experience at a given time and avoid the foregoing disadvantages, the technique and conventions of the life table have been developed, chiefly by actuaries in order to compute life-assurance premiums and annuities. But it should be noted here that the life table has great biological importance, quite unconnected with any computation of monetary values—for example, in the study of fertility and population. Until recently there were life-table studies of few animal species, but now the method is regarded as of increasing importance in ecological studies, and life tables have been constructed for many species. We shall consider here only applications of the human life tables for the Australian population to problems of medical or of general biological interest. It will be necessary to deal with some very general aspects of the technique of construction of the life tables, but the techniques of graduation (or "smoothing") will not be considered.

The Life Tables—Two Early Attempts.

It is assumed here that John Graunt (1620 to 1674) was the first, in modern times at any rate, to construct a life table, although the data on which he worked were extremely fragmentary. His life table is displayed in Figure II. Modern criticism suggests that his table presupposes an unduly high mortality in early adult life. Edmund Halley (1656 to 1742), the celebrated astronomer

and mathematician, in his studies at the end of the seventeenth century, realized that more adequate knowledge of the age at death in the population observed was necessary if tables were to be constructed from mortality rates. He therefore used an alternative method that can be appreciated only after the theory of the life-table population has been considered. Halley's life table is given in Figure II from figures cited by Greenwood (1942). The survivorship curve from a recent Australian life table is given for comparison.

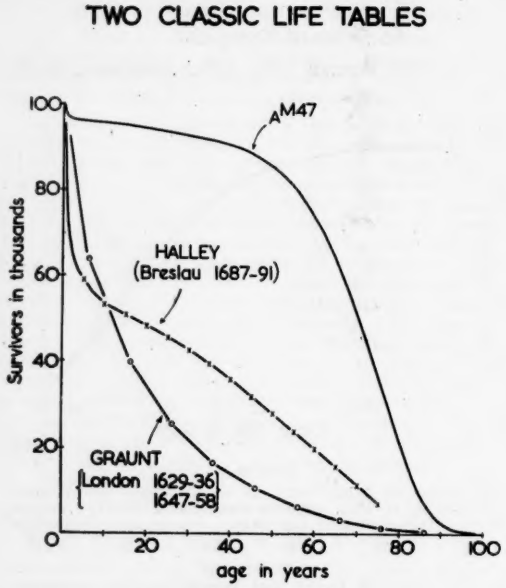

FIGURE II.

Two classical life tables compared with a modern life table (Australian males, 1947).

Th Fundamental Assumption of the Life Table.

The fundamental assumption of the life table is that it is possible to imagine a number of persons, 100,000 say, born and then submitted throughout their life to the same mortality rates as exist or have existed in some population at a given point of time. Another assumption is that the deaths in such a group of persons would not occur erratically, but would increase or decrease in a regular manner, even though this might need a very complicated mathematical formulation for description. There are no difficulties involved in this second assumption, but the first is quite arbitrary, even though it appears plausible. However, it is sound if the death rates in the observed population are stationary, as they were in England and Wales at the beginning of the last century.

The Selection of the Mortality Rates in an Actual Population.

Two methods of obtaining mortality rates have been used in constructing Australian life tables. For the earlier life tables constructed by the Bureau of Census and Statistics, Canberra, the deaths in the intercensal periods, 1881 to 1890, 1891 to 1900 and 1901 to 1910, were summed for the period of ten years. Estimates of the mean population in each of the corresponding age groups were obtained by means of the two censuses and statistics of births and deaths and migration. In 1921, however, it was decided to change the method of estimating the mortality rates to one whereby the deaths for the three years around the census were divided by three to obtain an average of deaths for one year, and this average number of deaths for each age group was divided by the population at the same age as enumerated at the census, which in every case occurred near the mid-point of the central year of the three considered. C. H. Wickens, Supervisor of the Census of 1911 in Australia and Commonwealth Statistician at the time of the 1921 census, points out (Wickens, 1930) that the change in the method was brought about as a result of three considerations: (i) The war had intervened in the period 1914 to 1918. (ii) The influenza epidemic had appeared in Australia in 1919. (iii) The mortality experience would apply approximately to a time with mid-point at least six years prior to the publication. In 1921 the mid-point would have been the year 1915 if computed on the old basis, and this at a time when mortality rates were decreasing rapidly. For the life tables constructed in conjunction with the 1933 and 1947 censuses in Australia, the same principle has been used. An additional reason for preferring the more recent method is that the census enumeration may be regarded as highly accurate, whereas the intercensal estimates, necessary in the computations by the older method, may be held to be less accurate.

An example of the value of the third of the considerations above was given in connexion with the 1933 Australian census, when the life table was prepared and ready for publication by February, 1936, the time of appearance of *Demography*, the annual bulletin of the Bureau, for 1934, and so less than three years from the mid-point of the experience considered. However, there are some reasons for preferring the older method. (i) Since more years are under consideration, the sampling errors are less. The avantage of reducing sampling errors by using a larger experience has to be weighed against systematic errors which may be introduced in the intercensal estimates. In Australia these systematic errors appear to be relatively small. (ii) The mortality rates over the longer period are more representative of an epoch, for in a shorter period it may happen that there is an epidemic of some size, or alternatively that such an epidemic as the influenza epidemic of 1919 has been deliberately avoided—so that the mortality figures are definitely "selected".

In conclusion, there is room for both types of life tables, and the older type is likely to become of increasing importance as the time from the mortality experience becomes greater. Life tables should also be constructed on the "generation" principle, by the use of the observed rates of mortality of generations born in, say, 1870, 1880 *et cetera*.

Select Life Tables.

The life tables constructed from the mortality experiences already noted in the last section have one feature in common—they describe the mortality of the whole population, there being no selection of persons. From time to time the life-assurance offices have published tables of the experience of assured lives—that is, of persons who have been accepted as risks suitable for life assurance. Since these persons are "selected", the select life tables, although showing what would be the experience of a group of initially selected or healthy persons, are of doubtful comparative value, as it can never be known whether the criteria of selection have remained constant over a period of time. Some life tables of great historical interest have recently been constructed by Peller (1949) from records of the ruling families of the old German and Central European States, and by Russell (1948) from mediæval English records.

The Mortality Rates in the Life Tables.

The fundamental step in the construction of a life table is to make the rates in the life table at every age equal to those in the actual observed population. To obtain the mortality rate in the life table, we note that if l persons are alive at the xth birthday and a proportion q die before the $(x+1)$th birthday, then only $(l-lq)$ will survive to the $(x+1)$th birthday. Unless the mortality rate is changing very rapidly at this time of life, then it may be assumed that on an average the lq persons who die will

each live one-half of a year. So that the mortality rate in the life table at any age is given by

$$m = \frac{\text{(number of deaths at that age)}}{\text{(number of years of life experienced at that age)}}$$

$$m = \frac{lq}{\{\frac{1}{2}(l+l-lq)\}}$$

$$m = \frac{2q}{(2-q)} \quad \ldots\ldots (1)$$

But there are usually a number of q's to compute and so they are distinguished as to age by means of a subscript, so the formula becomes

$$m_x = \frac{2q_x}{(2-q_x)} \quad \ldots\ldots (2)$$

By a slight rearrangement this result becomes

$$q_x = \frac{2m_x}{(2+m_x)} \quad \ldots\ldots (3)$$

So that the probability of a person aged x years exactly dying before attaining his $(x+1)$th birthday, or on the other hand the probability of surviving to his $(x+1)$th birthday, can be computed. The notation of the life table may now be formally introduced, with the remark that it is redundant in the sense that, given the number of persons beginning life in the life table and any set of life-table elementary values (for example, any set of the l_x, q_x, p_x or d_x for all integral values of x), the whole life table can be computed.

The Notation of the Life Table.

Probably nothing has done more to impede the study of the life table than the formidable notation developed by the actuaries. However, it cannot conveniently be studied without the use of six symbols, which are now introduced.

The Survivors, l_x.

The survivors after x years exactly will be denoted by the symbol l_x; l_x are the living after the elapse of x years. l_0 is commonly defined as being 100,000, so that usually 100,000 individuals are assumed to start life at age 0 years.

The Deaths at Age x of the Life Table, d_x.

There are l_x survivors to x years exactly and l_{x+1} to $(x+1)$ years exactly, so that there must have died $l_x - l_{x+1}$ between the xth birthday and the $(x+1)$th. This quantity, the number dying in the $(x+1)$th year of the life table, is d_x. So that,

$$d_x = l_x - l_{x+1} \quad \ldots\ldots (4)$$

Further,

$$l_x = l_{x+1} + d_x = d_x + d_{x+1} + l_{x+2} = \sum_{t=0}^{t=\infty} d_{x+t} \quad \ldots\ldots (5);$$

that is, where t can take integral values from 0, 1, 2, 3... up to the extinction of the table and the death of the last survivor, ∞ being used as an indefinitely large integer.

The Probability of Surviving One Year, p_x.

The probability of surviving from x years exactly to $(x+1)$ years exactly will be given by dividing the number surviving to the $(x+1)$th birthday by the number beginning the year, that is, attaining their xth birthday. This probability of surviving is denoted by p_x. In the notation above

$$p_x = \frac{l_{x+1}}{l_x} \quad \ldots\ldots (6).$$

The Probability of Dying Before the Next Year Elapses, q_x.

The probability of an individual aged x years exactly dying before he reaches $(x+1)$ years exactly is evidently a probability complementary to the probability p_x, since the individual alive at his xth birthday must either die before or survive until his next birthday. We define the probability of an individual aged x years exactly dying before he attains the age of $(x+1)$ years as q_x. Therefore,

$$q_x = \frac{d_x}{l_x} \quad \ldots\ldots (7).$$

It follows from (4), (6) and (7) that $q_x = \frac{(l_x - l_{x+1})}{l_x}$

$$= 1 - p_x \quad \ldots\ldots (8).$$

It is clear that the l_x, d_x, p_x and q_x are all dependent on one another and that, given one of them, all the others can be readily determined by simple arithmetic.

The Expectation of Life, $\mathring{e}_x$.

A measure that has an immense popularity in journalistic circles is the expectation of life, denoted by the symbols e_x or $\mathring{e}_x$. The complete expectation of life is the mean number of years that persons, aged x years exactly now, may expect to enjoy before death. Expectation is here used in a restricted mathematical sense introduced from the theory of gambling. If we are given a shilling every time a head appears but nothing for a tail, then the expectation of a single toss of a true coin is said to be sixpence. The years of life lived above age x years exactly are made up of two parts, firstly whole years of life and secondly the fraction of the year that each person will live in the year he dies. In the year commencing with the xth birthday, the group between them will live l_{x+1} completed years. The total number completed of years lived will then be the sum of the l_x from $(x+1)$ up to the termination of the table. Each person dying may be expected to live on an average one-half of the year in which he dies. The total number of fractional years lived will then be half the sum of the d_x from the year x up to the termination of the table, but this is only $\frac{1}{2}l_x$. So that

$$l_x \mathring{e}_x = \sum_{t=0}^{t=\infty} \{ t d_{x+t} + \tfrac{1}{2} d_{x+t} \} \quad \ldots\ldots (9)$$

$$l_x \mathring{e}_x = \sum_{t=1}^{t=\infty} l_{x+t} + \tfrac{1}{2} l_x \quad \ldots\ldots (10)$$

$$\mathring{e}_x = \tfrac{1}{2} + \frac{\sum_{t=1}^{t=\infty} l_{x+t}}{l_x} \quad \ldots\ldots (11)$$

The Mortality Rate in the Actual Population Observed, m_x.

The mortality rate of persons aged x years last birthday in the observed population is m_x. Thus at a census the persons, say males aged eleven years last birthday, will be enumerated. This number will be taken to be the mean number of persons aged x years last birthday alive in the population throughout the year. There will be in the year of the census some deaths of males aged eleven years. The mortality of males aged eleven years will then readily be obtained by division, so that

$$m_x = \frac{\text{deaths of persons aged } x \text{ years in a calendar year}}{\text{mean number of persons aged } x \text{ years alive during the year}} \quad \ldots\ldots (12),$$

x being in this case 11.

Construction of a Life Table.

It is not intended to discuss here the technical details of life table construction. A classical account is that of Henderson (1915), but possibly the simplest and most satisfying for a medical reader is that given by Dublin and Lotka (1936) in their monograph "Length of Life". Another recent description is given by Greville (1946) for the life tables of the 1940 census of the United States.

of America. It is sufficient to note in passing that usually some process of graduation is used and that a series of values for q_x and hence p_x is obtained. Every value of l_x can then be obtained by the formula (6), above, in the form

$$l_{x+1} = l_x \, p_x \quad \ldots\ldots\ldots\ldots\ldots\ldots (13).$$

Australian Life Tables.

The first life table in which Australian mortality experience was used was constructed by Pell (1867). Subsequent life tables, constructed by bodies other than the Bureau of Census and Statistics, Canberra, have been rendered superfluous by the publication, in the statistician's report on the Australian census of 1911, of life tables for the separate colonies and for Australia, based on mortality for the years 1881 to 1890 and 1891 to 1900, and of Australian life tables for the years 1901 to 1910. In the report on the census of 1911 (Knibbs, 1917), the data on which the life tables were founded were given in great detail. This practice had since 1917 been temporarily abandoned. For example, in the report on the 1921 census a short account of the technical details of making the tables was given, but no particulars as to the deaths and death rates. A further review of Australian life tables was given by Wickens (1930) containing complete references to all preceding Australian life tables. The life tables of 1933 were not accompanied by any detailed description, but reference is made in the census report to a special article in the "Official Year Book of the Commonwealth of Australia", Number 29, for 1936, in which, however, the additional details are concerned only with graduation minutiæ. These later practices, brought about doubtlessly in the interest of economy, must be regarded as unsatisfactory. A return to the older practices has been made by the publication of "Australian Life Tables, 1946-1948" (Balmford, 1950).

The Interpretation of the Life Table.

The Survivorship Values, l_x.—The figures for the life tables of the 1947 census have been published in a census bulletin (Balmford, 1950). By the use of these data the graph of l_x, displayed in Figure III, has been drawn and Table I compiled.

TABLE I.

Australian Life Tables. Comparative Number of Survivors (l_x) from 100,000 Born.

Age. (Yrs.)	1881 to 1890.	1891 to 1900.	1901 to 1910.	1920 to 1922.	1932 to 1934.	1946 to 1948.
			Male Lives.			
5	81,281	84,047	87,585	90,283	93,887	96,037
20	76,630	80,349	84,493	87,697	91,797	94,562
50	55,606	62,014	68,221	74,330	81,061	85,946
70	27,291	32,134	38,275	44,332	50,086	52,230
90	1328	1501	1652	2141	2935	3144
			Female Lives.			
5	83,090	85,842	89,285	92,148	94,993	96,851
20	79,099	82,473	86,459	89,906	93,341	95,953
50	59,978	66,095	71,945	78,313	83,680	89,011
70	34,877	40,943	46,793	54,771	59,629	65,398
90	2095	2833	3566	4238	5808	6556

For the males, beginning life with a group of 100,000, l_0, there is an immediate drop due to the infantile mortality to an l_1, of 96,801. The graph of l_x then becomes flatter, since, as we can see from the mortality rates in the actual population, the value of q_x reaches a minimum about the age of eleven years. After this the curve becomes steeper, gradually up to the early thirties, but then more and more rapidly. The greatest slope occurs at the ages around seventy-five years. The curve then becomes less steep and finally comes to lie along the horizontal axis, for there are few people left at these ages. After the age of 105 years there are virtually no survivors. Several points of interest arise from a study of the l_x curve, as follows.

1. The median age at death for males at the 1946-1948 rates of mortality in the life table was approximately seventy-one years. We may also obtain the ages at which there were 25% or 75% survivors. We have given these in Table II for six Australian life tables.

TABLE II.

Australian Life Tables. Age at Various Survival Rates as Deduced by Linear Interpolation from Australian Life Tables.

Proportion Surviving.	1881 to 1890.	1891 to 1900.	1901 to 1910.	1920 to 1922.	1932 to 1934.	1946 to 1948
			Males.			
75%	22·8	31·3	41·3	49·1	56·3	59·5
50%[1]	55·0	60·1	64·2	67·3	70·1	70·8
25%	71·3	73·6	75·4	77·6	79·0	79·2
			Females.			
75%	28·1	36·0	45·1	54·7	60·4	64·9
50%[1]	60·0	64·7	68·3	71·9	73·8	75·7
25%	75·4	77·3	78·6	80·8	82·2	83·0

[1] The 50% survivorship age is often spoken of as the "*vie probable*", which is not a good name. It may be called the "median length of life".

2. The modal age at death can be determined by the point of greatest slope of the l_x graph; but it is better determined from the d_x curve. For the 1947 tables we find these greatest slopes at the first year of life and in the last five years of the late seventies for males.

3. The upper limit of the life span in actual populations has usually been conceded to be approximately 105 years, although occasional persons do live beyond it. In the Australian life tables of 1933 the actuary assumed that this was the true upper limit and took q_{104} to be unity for both sexes. In the 1947 tables the actuary has used extrapolation methods to obtain the values of q_x above age eighty-seven years.

TABLE III.

Australian Life Tables. The Age at which the Second (Relative) Mode for Deaths Occurs in the Life Tables.

Years to which the Life Tables are Related.	The Age (Years) at which d_x is a Maximum.	
	Males.	Females.
1881 to 1890	68	77
1891 to 1900	75	77
1901 to 1910	74	75
1920 to 1922	77	79
1932 to 1934	78	79
1946 to 1948	75	80

4. A weakness of the l_x curve used as the means of comparison between two populations is that the slope of the graph gives a measure of the deaths *per annum* of the group of persons and not the death rates. This can be overcome by plotting the l_x logarithmically. The slopes of the curves plotted in these two ways are given by $\frac{dl_x}{dx}$ and $\frac{d \log_e l_x}{dx}$ or $\frac{1}{l_x} \frac{dl_x}{dx}$ respectively, in the notation of the differential calculus. This second quantity is referred to as "the force of mortality" or the instantaneous death rate—that is, the deaths per unit of time as the time of observation is made small. The concept of the force of

mortality is analogous, mathematically, to the force of interest in accountancy and the force of friction in physics.

5. The point raised in section 4 is important if the mortalities of two countries are being compared or the mortalities of two different epochs in the same country, for if there is initially a high rate of mortality in one population, then this fall will tend to conceal high rates of mortality in later life if the observer is using the slope of the l_x as a measure of mortality.

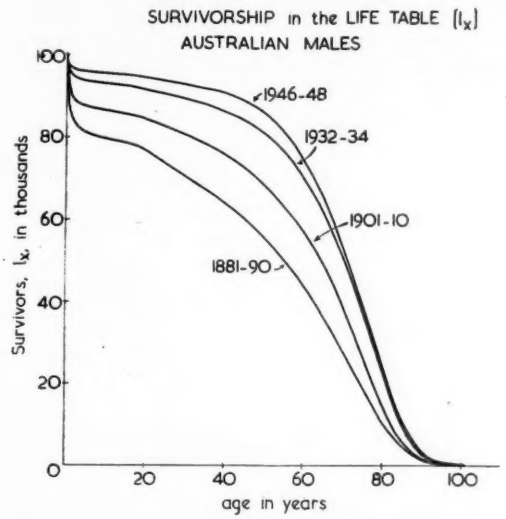

FIGURE IIIA.

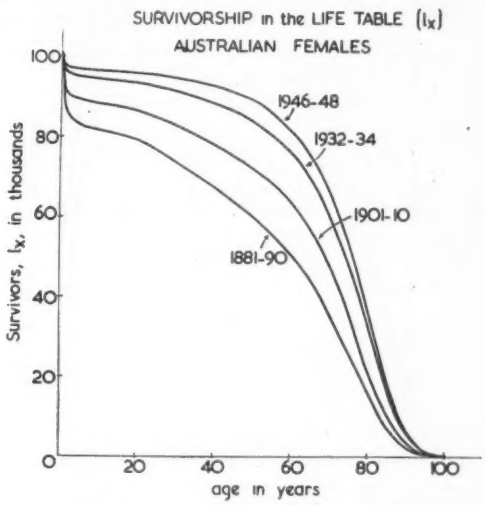

FIGURE IIIB.

A comparison of the expected survivorship for mortality rates prevailing in Australia; the l_x curves.

The Deaths by Age in the Life Table, d_x.—In Figure IV the values of d_x, the deaths at age x in the life table, have been plotted against the age. It will be noted that there is a mode for deaths for males in the first year of life that is, more deaths occur in the first year of life than in any succeeding year. A second relative mode occurs at the age of seventy-five years. Tracing out the features of the graph of d_x, we find that after the maximum in the first year of life the deaths in each succeeding year decline to a minimum at age eleven years and then rise fairly rapidly up to the age of twenty years. In the twenties the d_x are practically constant (d_x has a relative mode at age twenty-two years for the males in the 1947 life table), but then in the thirties begin to rise with greater slope to the graph. The rise is steady up to the age of seventy-five years, where there is a maximum as has already been noted. There is then a steady fall until the termination of the life table at age 105 years. The deaths, it will be noted, are given by the product of the l_x and the q_x, as can be seen from the definition of q_x in (7): $d_x = l_x q_x$. So that although the q_x is rising steadily throughout the latter part of life, there are fewer and fewer survivors, l_x, at each age, and as a result d_x has a maximum value not at the termination of the table but at a point much earlier, seventy-five years in the 1947 Australian life tables. The rate of change of the q_x with age is so rapid that the relative maxima in later life of the actual population and of the life table agree fairly closely. Thus the relative maximum in the years 1932 to 1934 in both sexes in the actual population was in the late seventies, its actual position in either case being somewhat obscured by the biases in giving ages, for it is a common experience that 0 and eight are preferred to seven and nine as the final digit when persons state their ages. It is of interest to note that this modal age at death has changed very little either in the actual population or in the life-table populations, with the single exception of the males in the 1881-1890 life table. In general it is to be noted that the female mode occurs a few years after the male, which is in accord with the more favourable mortality rates of the female at every age. In Australian life tables prior to those of 1947 an absolute mode of d_x has always occurred in the first year of life. With the coming of low infant mortality rates the relative mode in the seventies of the earlier life tables is replaced in the 1947 tables by an absolute mode for the females at age eighty years, and the relative mode of $d_x (d_{75} = 3067)$ is only slightly below the d_0 of 3199 for males.

The Interpretation of the p_x and q_x.—Since p_x and q_x are complementary probabilities, we need to consider only one of them, q_x. In actuarial circles and in the official publications an extended form of the p_x is used, the probability of surviving ten years. I myself consider that this is an unsatisfactory type of measure and of little value for comparison. To avoid the zeroes of the decimal values of q_x it is customary to use the value of 1000 q_x. These values have been plotted in Figure V for the 1947 life tables. The graph of q_x has a distinctive form. It has a high initial value of q_x, q_0 being the infantile mortality rate; q_x then attains a minimum at the age of approximately eleven years and then rises with increasing slope up to about the age of twenty years; through the twenties the rise is more gradual (in fact, in the 1947 life table for males there was a maximum at age twenty-two years and a second minimum at age twenty-five years); but then the rate of increase becomes more rapid and the value of q_x climbs steadily and rapidly through the remainder of the life cycle. Figure V is plotted on an arithmetic scale, but there is some advantage in plotting the q_x on a logarithmic scale. When this is done, as in Figure VI, the points from the age of fifteen years or so lie practically on a straight line, in general accordance with the Gompertz law that the rate of mortality increases geometrically with age or that the logarithm of the rate of mortality increases linearly with the age. No satisfactory explanation can be given of this law, but it graduates efficiently the life table experience of many countries. Such a graduation for the Australian males life table of 1933 is shown in Figure VII. It is seen that the fit of the graduation is excellent for ages above five years. It is probable that the most satisfactory way of comparing two life tables is to plot the q_x

of each on the same graph logarithmically against an arithmetic scale for the ages as has been done in Figure VI.

The Complete Expectation of Life at Ages, $\mathring{e}_x$.—At any age, with the aid of the life table, the number of years the

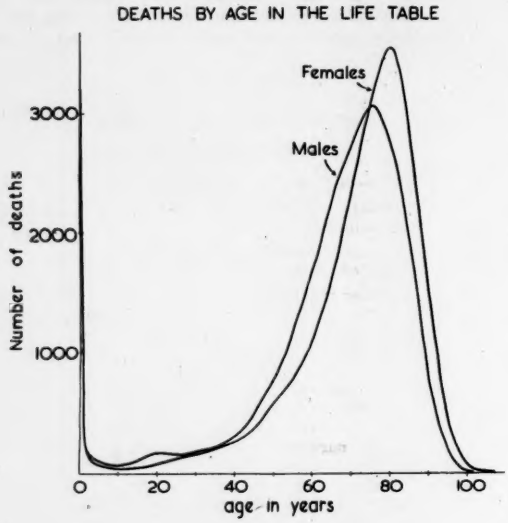

FIGURE IV.
The deaths by age in the life tables, Australia, 1947.

survivors may be expected to live between them and hence the mean number of years that the survivors at age x years may be expected to live can be computed, as has already

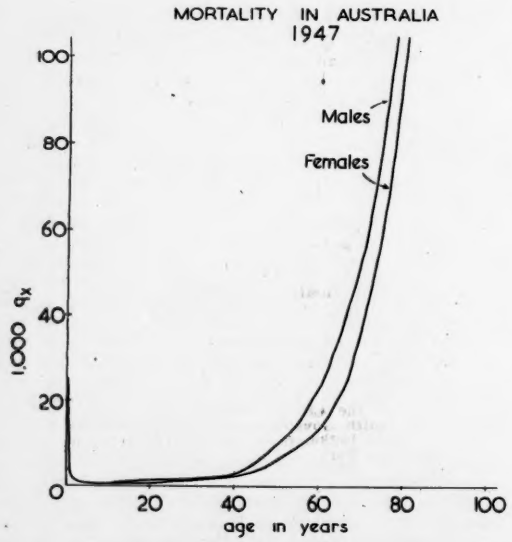

FIGURE V.
The change in death rates with age in Australia, as measured by $1000q_x$. Arithmetic grid.

been explained. This mean value is the complete expectation of life, usually briefly referred to as the expectation of life. In order to compute this expectation of life we make the usual assumption that the deaths occur equally spaced throughout any single year of life of the life table; thus it is assumed that the persons dying between their xth and $(x + 1)$th birthdays die aged $(x + \frac{1}{2})$ years on an average. This assumption is sufficiently accurate at every age except in the first year of life, when various formulæ have been devised to allow for the great bulk of the deaths occurring early in the year. We might equally well determine this mean length of life lived by those dying in the first year of life by consulting the original statistics of the deaths on which the particular life table has been

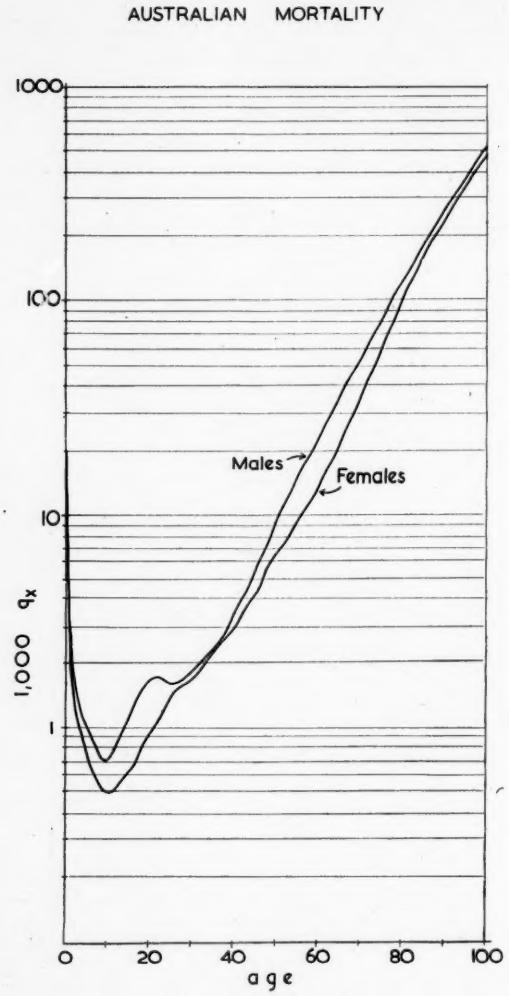

FIGURE VI.
The life-table mortality rates for males and females compared for the 1947 life tables. Semilogarithmic grid.

founded. For elementary work this refinement is unnecessary. With this exception then the expectation of life may be computed by the use of the formula

$$\mathring{e}_x = \frac{\text{(years of life lived by persons above age } x \text{ years in the life table)}}{\text{number of persons attaining age } x \text{ years}} \quad (14).$$

The graph of the expectation of life for males in the 1947 males life tables is as follows. At birth the expectation of life for males is 66·1 years approximately; the expectation of life then rises at the age of one year to a maximum of

67·25 years, and then falls steadily to the end of life practically in the form of a straight line. It may be thought strange that children aged one year are expected to live longer than children at birth. But a little reflection shows that it is a mean that is being measured, and that some 3·2% of male infants die in their first year of life

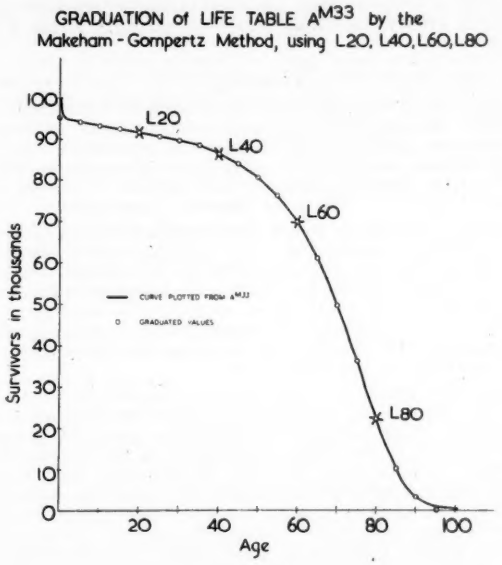

FIGURE VII.

The male survivorship table for Australia, 1933. Graduated by the Makeham modification of the Gompertz method.

and so bring down the average expectation of life. In actuarial terms this phenomenon of the expectation of life increasing with the age may be expected to occur wherever

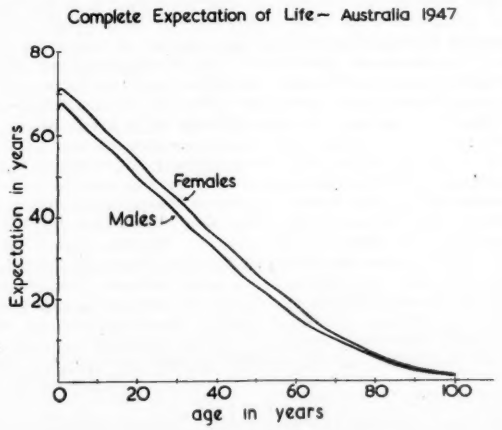

FIGURE VIII.

The complete expectation of life, Australia, 1947.

the force of mortality is greater than the reciprocal of the expectation of life of the survivors. In some animals it is possible to imagine a condition in which the chance of dying in any small unit of time is a constant over a wide span of the life cycle; under these conditions the expectation of life will remain a constant over a large part of the life span. It will also be noted that the slope of the curve of $\mathring{e}_x$ would be exactly 1·0 were all the deaths in the life table to occur at the same age. As deaths occur throughout life the slope is never 1·0, but in fact in the Australian life tables was close to 0·9 over the ages four to fifty-four years. After this age the slope tends to diminish.

The expectation of life has been widely used as a measure of hygienic conditions, sometimes in the form of its reciprocal, the life-table death rate, or true death rate. Although it gives a good summary of mortality experience, any standardized death rate may be used. Dublin and Lotka (1937) showed that there was a high degree of correlation between the life-table death rate and the death rates standardized on to a standard million (England and Wales census population of 1901).

The Comparison of the Australian Life Tables of 1946 with Preceding Australian Life Tables.

Balmford (1950) has made in his paragraph 24 the following observations:

(*a*) A further substantial reduction has occurred in the mortality rates in the first year of life.

(*b*) A further marked improvement is evident in the vitality of both males and females up to about age 40.

(*c*) A less pronounced improvement is shown in the mortality rates of both sexes between ages 40 and 80; indeed, in the case of males, the rates between ages 60 and 80 are generally in excess of those recorded in the 1932-34 Tables.

(*d*) Because of the different methods of graduation, referred to in paragraph 19, no valid conclusions can be drawn from comparative tables as to the trend in mortality rates from age 80 onwards; the indications are, however, that, if the methods of graduation had been the same, the new mortality rates for males would have been somewhat higher than, and those for females approximately equivalent to, the mortality rates derived from the 1933 Census.

(*e*) Although the 1932-34 Tables disclosed that the mortality rates of females at the child-bearing ages close to age 30 were slightly in excess of the rates for males at the same ages, the differences in the rates on this occasion are slightly in favour of the females.

(*f*) The mortality rates for females are lighter than those for males at all ages.

(*g*) On the whole the vitality of the female population shows a greater degree of improvement over the last fourteen years than does that of the males.

Summary.

The principles of the life tables have been discussed. Brief mention has been made of some classical life tables. The elementary functions of the life table have been defined. Special reference has been made to the 1947 Australian life tables.

Acknowledgements.

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PRE-BRONCHIECTASIS.[1]

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THE condition about which I am going to speak is what has been called pre-bronchiectasis or potential bronchiectasis. The pre-bronchiectatic state is one in which the patient, if not vigorously and correctly treated, will possibly develop bronchiectasis, or in which there are early signs of bronchiectasis but these changes are still in a reversible state. After a resume of the literature I will give an analysis of 50 cases of pre-bronchiectasis.

Although bronchiectasis was first described by Laennec in 1823, a perusal of the old and not so old hospital records shows that very little interest was taken in it until practically the last two decades. No doubt the use of radio-opaque fluids during the last quarter-century has been one of the factors which began to stimulate interest, and recently a voluminous literature has appeared on the subject. In the 1930's this was mostly given to the ætiology and pathology, while in the late 1930's and in the 1940's despite the diversions of a world war, a large literature accumulated dealing with the treatment. While this dealt, latterly, with treatment by the antibiotics, the main trend of the literature has been on the subject of surgical treatment by lobulectomy, lobectomy or pneumonectomy. There is left, however, a large group of patients who are not suitable for surgical measures, but who require constant palliative treatment, which may include some form of chemotherapy. Patients of this type have established bronchiectasis, and I have already dealt with their treatment in previous papers (Harwood, 1945, 1946).

Cookson and Mason in 1938 published a paper entitled "Bronchiectasis—A Fatal Disease". This blunt title should surely have struck us forcibly and aroused us to the full import of tackling bronchiectasis—and surely the best way to tackle it is by trying to prevent it. Lander and Davidson (1938) strongly emphasize their opinion that the above-mentioned pessimistic view is hardly justified by the facts. Still, the recognition of the pre-bronchiectatic state and its treatment must be one of the most important aspects of dealing with bronchiectasis.

Amongst the full literature quoted above there is little dealing with preventive measures. Bronchiectasis is a very common condition, which should not be so if the prospective cases are dealt with early and correctly. There are, of course, those cases which should be prevented by the recognition that some foreign body is present requiring removal, and also those cases of post-operative atelectasis (Harwood, 1947) in which bronchiectasis may easily develop if urgent and correct treatment is not given. It is not this type of case we are dealing with here.

In 1934 Anspach drew attention to X-ray findings in cases in which a cough was present which had failed to settle in a reasonable time after some pulmonary infection. Fergurson (1937) and Hollinger (1938) mentioned the prevention of bronchiectasis, as also did Watson and Kiblin in 1938, and later Hinshaw and Schmidt (1944); but in spite of this no great interest has apparently been taken in this side of the subject by those who most frequently come into direct contact with these pre-bronchiectatic patients—namely, the general practitioners. One still finds that these pre-bronchiectatic patients are more likely to be referred for treatment by patients or ex-patients of the bronchoscopic clinic than by outside doctors with, I should like to say, a few notable exceptions. Recently Badger (1947), Palley (1948) and Elaine Field (1949) have again brought up the subject of prevention, while Chevalier Jackson (1950) has once more most strongly stressed its importance.

Pathology.

Much has been written on the causes of bronchiectasis, and the final opinions appear to centre around bronchial obstruction, primarily, with infection secondarily as the main factors, together with atelectasis as a sequela of the obstruction. Certain factors which are the cause of the obstruction are of the most extreme importance.

Firstly, there is the size of the bronchi, which in children are extremely small and are therefore easily blocked. Jackson points out that a millimetre of mucosal swelling all round the wall of a four-millimetre bronchus will reduce the lumen to two millimetres, while the same amount of swelling in a two-millimetre bronchus will cause complete obstruction. The smaller the bronchus, the more liable it is to obstruction and obliteration.

Secondly, there are the type and amount of secretion present. Practically all pulmonary infections produce some secretion. This secretion is always a potential hazard, because if it is not removed promptly it may occlude the airway. Inflammatory purulent (Woodwards and White, 1949) and mucopurulent secretions are extremely adhesive. If they are allowed to stagnate and thicken into masses they become obstructive endogenous foreign bodies, difficult to expel. Removal of these plugs is as important as the removal of any other foreign body.

Thirdly, comes the very common accompanying allergy. Watson and Kibler found in fully 90% of cases of bronchiectasis a diagnosis of allergy, while Elaine Field found that of pre-bronchiectatic children 42·7% had symptoms suggestive of asthma, against 33·1% with asthma in established bronchiectasis. Certainly a history of allergy is common. The slightly swollen lining membrane with its thick secretion will help to cause obstruction in the bronchi.

Histologically the changes in the bronchial wall may not be so pronounced as those described by Erb (1933), who suggested that ulceration and destruction of the bronchial wall were essential factors in the development of bronchiectasis. Lee Lander and Davidson, in a study of lobectomy specimens, have shown the presence of intact mucosa and even the presence of cilia, though with areas of metaplasia and hyperplasia, and in many cases all the normal layers of the bronchial wall were present. In some of their specimens infection was shown only by some slight degree of round-celled infiltration. Areas in which bronchial wall structure was not seen were saccules. These, though lined by epithelium, were surrounded by fibrous tissue; but being found in the periphery of the lung they are probably caused by the extreme dilatation of minute bronchioles which normally contain no glands or cartilage and practically no muscle. Robinson (1938) also found that in the more acute stages there might be no pronounced destruction of the cartilage and that cilia might be present, and pointed out that from his studies infection appeared to be a main factor.

This finding—that the pronounced changes suggested by Erb need not be present—would seem to be borne out clinically by the following case, in which the condition was shown by X rays.

A girl, aged twenty-two years, with a prolonged history since pneumonia at the age of two years, had had a chronic cough with a moderate amount of sputum for many years, and cylindrical bronchiectasis in the lower lobe and lingula of the left lung. Her condition cleared up after fourteen months of bronchoscopic treatment only. Her later bronchogram reveals no bronchiectasis, and she now has no sputum either on coughing or on lavage.

[1] Read at a meeting of the Oto-Laryngological Society of Australia (British Medical Association) on June 4, 1951.

This would appear to indicate the inadvisability of diagnosing irreversible bronchiectasis in lesser lesions, no matter how long the history, until efficient bronchoscopic drainage and treatment have been given for at least twelve months from the time of diagnosis of the bronchiectatic lesion.

Elaine Field considers that reinfection is a predisposing factor to permanent changes, but states that as an essential factor it is still unproved. However, infection of the bronchial wall with destruction of the supporting elements and later fibrosis renders the wall more susceptible to pressure changes within the chest, and these pressure changes are a determining factor in the dilatation.

Anspach, Lander and Davidson, and Hollinger and others, stress very strongly the essential factor of a certain degree of atelectasis as an antecedent to dilatation of the bronchi. This must, of course, be caused by some degree of obstruction.

Any condition interfering with the free flow of air from the bronchi to the alveoli will cause collapse of the lung beyond the site of obstruction. This is common in children, in whom the typical respiration is mainly abdominal rather than thoracic. Once obstruction has occurred in these children there is less likelihood of its being relieved with this type of respiration.

Adams and Escudero (1938) have shown by experiment that obstruction was incomplete in every instance in which bronchiectasis was produced. Bronchiectasis was never found with complete obstruction. Clinical and experimental evidence points to bronchiectasis as being primarily due to interference with drainage by some form of obstruction damming back retained secretions. Hence (Clerf, 1932) the recognition of bronchial obstruction is extremely important, as however small the bronchus, irreparable damage may be done if the obstruction is not relieved.

The commonest predisposing causes of bronchiectasis (Raia, 1938) are pneumonia or bronchopneumonia secondary to whooping-cough, measles, pink disease *et cetera*, in which the sticky and glairy secretion, aided by the hypertrophy of the mucosa in allergic subjects, will easily cause obstruction of the smaller bronchi. If the secretion is sufficiently liquid, movement, cough, posture *et cetera* will expel it; but if this does not occur a piece of inspissated mucus becomes lodged and will cause obstruction to the airway and some degree of collapse.

While some of these cases resemble pneumonia, radiological findings may demonstrate that the process is one of atelectasis. If the condition in these cases settles down, the shadows will clear up completely. The reversibility (Fleischner, 1941) of bronchiectasis in the early stages is now an established fact, so that the problem, as has already been mentioned, is to recognize these early reversible cases and provide the patients with suitable antiatelectatic and respiratory treatment in order to avoid as far as possible the well-known later severe damage. As long as there is a chance for the alveoli to be reaerated, then the condition is reversible whether infection has occurred or not. In these pre-bronchiectatic cases the plain X-ray findings often consist of increased radial streakings especially at the lung bases, and during exacerbations these may vary in intensity.

There are two main groups in these pre-bronchiectatic or potential bronchiectatic cases, as follows. (i) Those in which everything suggests bronchiectasis in the history but in which we find that the bronchogram is normal. Watson and Kibler suggest that these cases may be even of ten years' standing. This has been confirmed in my series. (ii) Cases in which the histories are similar but minimal signs suggesting bronchiectasis are seen in the bronchogram. These signs may be very slight and localized to an isolated group of small bronchi, and may be dilatations or areas of atelectasis. These dilatations are not the temporary dilatations seen after pneumonia, which clear up quickly if no obstruction is present. Often pulmonary collapse may be shown in a bronchogram by the appearance of some crowding of the bronchi.

X-ray and clinical evidence of nasal sinusitis is very common in these cases, as in those of established bronchiectasis.

Symptoms.

It has been shown that 65% of all cases of bronchiectasis develop in the first two decades of life—that is, when the patient usually suffers from the infections which so frequently precede the development of this disease. Hence it is amongst young children that most of the preventive work must be carried out. What I look on as a pre-bronchiectatic patient is one who has had whooping-cough, measles, bronchopneumonia, pink disease or pneumonia, and has been left with a cough, not always productive, for eight to ten weeks after the infection—particularly when, added to this, nasal discharge is present and the patient is allergic or has an allergic family history.

As has already been stated, some of these patients give a much longer history of cough than this, but may still be in the pre-bronchiectatic class. However, it should be regarded as very unwise to leave them for longer than eight to ten weeks before starting efficient treatment, as they may rapidly pass to an irreversible state.

It must be remembered that in a young child a productive cough may not produce any obvious sputum. While sputum is usually expectorated after the age of eight years, the younger child more often swallows it, and under the age of three years this is almost universally so.

There may also be low-grade fever, and physical signs may be present at the lung bases. The masking effects of sulphonamides and the antibiotics on pulmonary infection are well recognized and should not be overlooked.

All these patients should have an X-ray examination of the chest and nasal sinuses; but if the X-ray findings are negative the history of the patient still demands correct, immediate and vigorous treatment.

In the cases of longer standing there may be loss of appetite, disinclination to play and retardation of the normal progress of the child.

The symptoms of potential borderline involvement may simulate those of bronchiectasis very closely, as also may the X-ray findings in the plain X-ray film.

Treatment.

Early and complete drainage of the bronchi is essential if the development of bronchiectasis is to be avoided. Anspach has shown clearly that if early pre-bronchiectatic patients are left untreated they will eventually develop typical bronchiectasis. Fleischner (1949) has also shown a return to normal of dilated bronchi in early cases, with subsidence of the parenchymatous changes which caused the mechanical disturbance. It has also been shown that in cases in which drainage was established early by one or two bronchoscopic aspirations, and in which the inflammatory stenosis was comparatively short, subsequent bronchographic examination revealed a normal bronchial tree.

The preventive measures should, naturally, start with the better treatment of the predisposing causes, particularly bronchopneumonia, pneumonia, whooping-cough, measles and so-called chronic bronchitis in children. Attempts may be made to modify the disease itself—as, for example, by pertussis injections and the efficient use of chemotherapy.

Convalescence after these conditions should be very carefully watched. Breathing exercises may help considerably, and these may be begun immediately after the acute stage has subsided. Play in the sunshine and fresh air, with adequate rest periods to prevent overtiredness, is always indicated, as this is a natural stimulus to deep breathing, which acts as one of the best preventives of bronchial obstruction.

The general health should be built up with a balanced diet of high vitamin content, as collapse will occur more readily in the debilitated child with poor respiratory movement. Great care must be exercised in avoiding exposure to infections and in prompt treatment of even a mild cold.

Any upper respiratory tract infections should be treated energetically, as sinus infection acts as a focus from which lung infection may follow a collapse. If the tonsils and adenoids are grossly infected it may even be considered advisable to remove them.

In all cases there should be a follow-up examination and if any cough is present after it should normally have been expected to subside, an X-ray picture should be taken, and if necessary this examination should be repeated.

If, in spite of this active convalescence, follow-up and treatment, the patient still continues to show signs suggestive of pre-bronchiectasis, even more active treatment must be instituted.

The use of inhalations to loosen secretions before postural drainage, postural drainage and chest clappings while the patient is being postured, and encouragement of the child to cough up secretions should be tried. If these methods do not show immediate results, bronchoscopic aspiration and lavage must be undertaken to remove the obstruction, which is usually inspissated mucus, in order to help reaerate the lung.

Opiates must definitely be avoided, and as Chevalier Jackson puts it, "their routine use as antitussives is one of the most deplorable and widely spread therapeutic errors in the history of medicine". He also strongly suggests that the incidence of bronchiectasis could be reduced to a relative rarity if every patient slowly recovering from acute infections of the lower part of the respiratory tract was promptly given the benefit of bronchoscopic aspiration. The thick, tenacious secretions occurring as localized obstructions, with which pulmonary drainage is unable to cope, must be removed by bronchoscopic aspiration. This aspiration will greatly help in the restoration of normal drainage by taking the load off the clogged cilia.

The chief therapeutic use of the bronchoscope is to establish a clear and adequate airway to the alveoli, and to reestablish normal physiological upward drainage by ciliary wafting, tussive squeeze and the blast of the cough itself.

The bronchoscopic picture in these patients is of extreme importance and interest. In some cases there may be a slight degree of generalized inflammation; in others the trachea and opposite main bronchus may be normal, while on the involved side the bronchus may be found to be considerably inflamed and to contain a moderate amount of thick, viscid mucopus. Aspiration reveals an œdematous stenotic bronchial orifice, which leads directly to the area shown by the X-ray film to be atelectatic. This orifice is usually intensely red and almost occluded by inflammatory swelling. Thick, tenacious mucopus may be seen exuding from the bronchus when the patient coughs. Obviously only a slight degree of mucosal inflammation together with this heavy mucopus is necessary to block the tiny bronchial divisions of a child.

A bronchogram is usually taken to demonstrate the extent of the lesion and repeated aspirations may be necessary.

Analysis of Cases.

I will now discuss 50 cases of pre-bronchiectasis, in all of which I believe irreversible bronchiectasis might have developed if bronchoscopic treatment had not been given. Some of you may ask what evidence there is that these children would have developed an irreversible lesion without treatment. That is rather a difficult but pertinent question, but I hope to convince you that this would probably have happened to most of these patients if they had not been taken in hand.

I will discuss 30 cases falling into the first group of pre-bronchiectasis—namely, those in which there was a suggestive history but a normal bronchogram—and 20 cases falling into group two—that is, those in which there were a suggestive history and slight dilatations or crowding of the bronchi as shown in the bronchogram.

Symptoms.

The history of both these groups is fundamentally the same as that of any established case of bronchiectasis. That is, after one of the usual predisposing causes the patient has developed a cough which has remained with him ever since and has been constant or occasionally intermittent. The most outstanding symptom in all cases was a constant cough.

Usually there was some nasal discharge as well, and it was common to find an allergic background. Frequent colds were often complained of, and tiredness and loss of interest in work and play. Loss of time at school gave a good indication of the severity of the case. The plain X-ray film usually showed an increase in the basal markings.

TABLE I.

Age of Onset of Symptoms.

Group.	Average Age when Symptoms Developed.	Average Age when Treatment was Commenced.	Duration of Symptoms Before Treatment.
Group I	2¼ years.	5¾ years.	3½ years.
Group II	3 years.	6¼ years.	3¼ years.

I would call the patient with the foregoing history and a normal bronchogram a pre-bronchiectatic patient. We can classify those patients who show minimal lesions in the bronchogram as pre-bronchiectatic only after we have seen the results of treatment—namely, the relief of symptoms and the proof by later bronchograms that the patient's lung fields have returned to normal.

TABLE II.

Predisposing Causes of Cough.

Group I.		Group II.	
Condition.	Number of Cases.	Condition.	Number of Cases.
Pertussis	12	Pertussis	8
Bronchitis	8	Bronchitis	4
"Cough developed" ..	4	"Cough developed" ..	3
Bronchopneumonia ..	3	Bronchopneumonia ..	3
After tonsil and adenoid operation	2	With osteomyelitis ..	1
Measles	1	Measles	1

I have suggested that all patients with persistent cough eight to ten weeks after the original infection should have a plain X-ray film of the chest taken. If the basal markings are increased, bronchoscopic treatment should be commenced at once. If basal markings are not increased it may be thought that one can wait another two weeks, but if symptoms are still continuing then I think that a bronchoscopic examination should no longer be delayed.

Only one patient in this series did come up for treatment ten weeks after the original infection of measles, and I would point out that this particular patient, while having normal appearances in a plain X-ray film, already had signs of early dilatation which took fifteen months to clear up completely. This case alone should be sufficient to justify early treatment in all suspicious cases, especially as only three other patients had clear lung fields in the plain X-ray film, and one of these also had early dilatation.

While 16 patients in the second group had a normal bronchogram after treatment, four did not have a follow-up bronchographic examination as the parents felt that the children were so well that a further examination was not warranted.

TABLE III.
Group I.

Case Number.	Patient.	Sex.	Age. (Years.)	Duration of Symptoms.	Report on Plain X-Ray Film.	Bronchogram.	Date of Last Bronchoscopic Examination.	Parents' Last Report.
1	G.S.	F.	7½	6 years.	Pronounced catarrhal changes at at base of right lung.	Not taken.	20. 8.42	7. 1.50: "Is extremely well. Has had no cough at all since 15.10.42."
2	D.B.	M.	7	2 years.	Gross chronic catarrhal changes at both lung bases.	No evidence of bronchiectasis.	13. 4.44	29. 4.50: "Has no cough. Rarely gets a cold and his health is very good."
3	R.O.	M.	4	2½ years.	Increased markings at both lung bases.	No evidence of bronchiectasis.	7. 1.47	8. 1.50: "Has been very well. Cough on only very rare occasions."
4	E.F.	M.	9	8½ years.	Clear.	No evidence of bronchiectasis.	8.11.45	7. 1.50: "Extremely well. Never looked back. Commented on by all the neighbours. Health marvellous."
5	K.C.	M.	2¼	2 years.	Patch of consolidation at right lung base.	No evidence of bronchiectasis.	23. 8.45	13. 3.50: "General health is very good indeed now. He has had no cough."
6	D.W.	F.	6	4 years.	Increased markings at both lung bases.	No evidence of bronchiectasis.	24. 7.47	24. 7.47: "Has no cough now and is very well indeed."
7	J.H.	F.	5	3 months.	Slight bronchitic changes at both lung bases.	No evidence of bronchiectasis.	10. 1.46	9. 1.50: "Greatly improved. Sleeps all night. Used to get up three or four times. All neighbours comment on change. Is 100%. Plays tennis and basketball."
8	J.S.	M.	7	3 years.	Pronounced chronic bronchitic changes at both lung bases.	No evidence of bronchiectasis.	10. 5.46	9. 1.50: "Is now 100%. Has no cough. Is doing well at school and sport. Occasional watery nasal discharge."
9	A.O'C.	M.	3	6 months.	Catarrhal changes of both lungs.	No evidence of bronchiectasis.	20. 3.47	11. 1.50: "Very well. Is big and strong and full of energy. No cough. Topped class at school.
10	R.K.	M.	12	10 years.	Catarrhal changes at both lung bases.	No evidence of bronchiectasis.	19. 6.47	11. 4.50: "Never has a cough now and never misses a day at school since treatment."
11	L.C.	M.	3	2½ years.	Slight increase in basal markings.	No evidence of bronchiectasis.	15. 5.47	8. 3.50: "Very well. No cough. Rides 3½ miles to school every morning and never misses."
12	M.D.	F.	4½	9 months.	Chronic bronchitic changes at both lung bases.	No evidence of bronchiectasis.	14.11.46	15. 3.50: "Extremely well. Never has a cough now."
13	W.S.	M.	5	4 years.	Dulness at right cardio-phrenic angle.	No evidence of bronchiectasis.	13. 2.47	15. 5.50: "Very well now and never has a cough at all."
14	A.T.	M.	7	8 months.	Patchy consolidation at right lung base.	No evidence of bronchiectasis.	15. 8.46	18. 5.50: "Has been very well indeed since the treatment."
15	K.B.	M.	3	2 years.	Chronic bronchitic changes at both lung bases.	No evidence of bronchiectasis.	10. 1.47	10. 1.50: "Very well indeed and only gets a very occasional cough."
16	W.E.	F.	4	6 months.	Slight increase in markings at right lung base.	Not taken.	5.12.46	12. 1.50: "Very well now Never has a cough."
17	L.M.	M.	5½	5½ years.	Increased markings at both lung bases.	No evidence of bronchiectasis.	4.12.47	11.10.50: "Has been extremely well. Never looked back. Doing well at school and all sport. Quite a different boy. No cough for three years."
18	D.D.	M.	7	2 years.	Considerable increase in markings at both lung bases.	No evidence of bronchiectasis.	22. 1.48	23. 1.50: "Extremely well. Friends remark about it. No cough. Doing very well at school and sport. Is now 100% better."
19	B.T.	F.	9	7½ years.	Increased peribronchial markings, especially at lung bases.	No evidence of bronchiectasis.	15.1.48	29. 3.50: "Has developed surprisingly since treatment. Never has any cough now."
20	D.B.	M.	2¾	2 years.	Increased markings at both lung bases.	No evidence of bronchiectasis.	19. 2.48	18. 1.50: "Is very much better. In fact has been a different child. Never coughs now."
21	J.W.	F.	8	7¾ years.	Slight bronchitic changes in both lungs.	No evidence of bronchiectasis.	22. 6.50	12. 1.51: "No cough. Very well indeed. Goes to school in all weathers. Used to be away alternate weeks. Doing well, eating well and is a different child in every way."
22	C.F.	F.	2	3 months.	Clear.	No evidence of bronchiectasis.	14. 4.49	29. 3.50: "Never has a cough and has never been so well."
23	R.S.	M.	2	2 years.	Increased markings at right cardio-phrenic angle.	No evidence of bronchiectasis.	24.11.49	1. 6.50: "Very much better but gets some asthmatic attacks still."
24	R.B.	M.	10	6 years.	Pronounced catarrhal changes, especially at lung bases.	No evidence of bronchiectasis.	1. 6.50	16.10.50: "Greatly improved. Has no cough and is very well indeed."
25	R.McK.	F.	4	1 year.	Slight increase in markings at cardio-phrenic angles.	No evidence of bronchiectasis.	10. 8.50	22.12.50: "Cough has all settled down now and she is a completely different child. Everyone remarks how changed she is."
26	R.B.	M.	4	3¾ years.	Pronounced bronchitic changes, especially at right lung base.	No evidence of bronchiectasis.	30. 8.50	23.10.50: "Very well. No cough at all. Is quite a different child."
27	F.D.	F.	12	4 years.	Increase in markings at both lung bases.	No evidence of bronchiectasis.	27. 7.50	31.10.50: "Very much better. No cough at all. Still has a little asthma."
28	D.K.	F.	7	4 years.	Catarrhal changes in both lungs.	No evidence of bronchiectasis.	16.11.50	30.10.50: "Has been completely different child. No cough. Everyone sleeps. Eats terrifically. Is most boisterous and we thank God when she gets to bed at seven o'clock. Was always very quiet."
29	J.H.	F.	7	1 year.	Increased markings at both lung bases.	No evidence of bronchiectasis.	2.11.50	2.11.50: "Has been extremely well. No cough. Sleeps and eats well. Still gets a little tired."
30	S.H.	F.	6	3 years.	Increased bronchial markings at both lung bases.	No evidence of bronchiectasis.	13.10.50	12. 2.51: "Has no cough at all and is extremely well. Has altered in every way. Even walks differently, and does not slouch. Came fifth in class."

The fact that in most of these cases the condition was so long established before the patients received bronchoscopic treatment, to which they reacted so well, must counteract any suggestion that sufficient time had not been allowed to elapse for the result to be known of other treatment of varying types which they had all received.

It will be seen that, as in established bronchiectasis, whooping-cough is one of the commonest predisposing causes, as we would naturally expect.

Environment.

It was found that in the two groups 17 patients were public hospital patients and 33 were private patients—that is, a ratio of 2:1, private to public hospital patients. From this it would seem fair to assume that poorer living conditions do not predispose children towards the development of pre-bronchiectasis.

Sputum.

Only 18 patients in Group I coughed up any sputum, and of these seven coughed up as much as four or five ounces daily. Three had been noticed to swallow it and the remainder had no obvious sputum. In Group II 17 patients coughed up sputum. However, the average age of the children in this group was a little higher than that in Group I.

Bacteriological investigations showed the usual flora found in established bronchiectasis — pneumococci, hæmolytic streptococci, non-hæmolytic streptococci, *Streptococcus viridans*, *Staphylococcus aureus*, *Hæmophilus influenzæ* and *Bacillus coli communis, et cetera*. These were found in both groups.

Hæmatological Findings.

The white cell count was slightly raised in an equal proportion of cases in both groups, as also was the sedimentation rate. In neither group was there any pronounced lowering of the hæmoglobin value. In Group I the value averaged 90%, while in Group II it averaged 89%.

TABLE IV.
Group II.

Case Number.	Patient.	Sex.	Age. (Years.)	Duration of Symptoms.	Report on Plain X-Ray Film.	First Bronchogram.	Second Bronchogram.	Date of Last Bronchoscopic Examination.	Parents' or Guardian's Last Report.
1	M.S.	F.	4	1 year.	Slight chronic bronchitic changes.	Early dilatation behind heart, left side.	No evidence of bronchiectasis.	30.11.50	30.11.50: "Very well indeed. No cough. Topped her class. Is quite a different child."
2	J.T.	F.	6	3 years.	General bronchitic changes, more pronounced on left side.	Small suspicious area in right cardio-phrenic angle.	No evidence of bronchiectasis.	7. 7.50	8. 7.50: "Parents recently died. Guardian says extremely well. No cough."
3	N.W.	F.	4	2½ years.	Increased markings suggesting bronchiectasis.	Very early bronchiectasis, left lung base.	No evidence of bronchiectasis.	16.11.50	15.12.50: "Has been very well indeed. No cough now."
4	P.McW.	F.	8	7 years.	Patchy dulness at both lung bases.	Suggestive of slight saccular dilatation.	Not taken.	27. 5.48	16. 3.50: "Very much improved. No cough."
5	J.W.	M.	9	4 years.	Bronchial changes at both lung bases.	Right lung base shows suspicious dilatation.	No evidence of bronchiectasis.	27. 7.50	3.11.50: "Very well. No comparison at all with what he was like. Fourth in class."
6	D.H.	F.	8	6 years.	Increase in basal markings.	Suggestive of early bronchiectasis at left lung base.	No evidence of bronchiectasis.	17. 3.49	25. 3.50: "Very well. Well developed and appears very healthy."
7	B.T.	M.	10	10 years.	Chronic catarrhal changes most pronounced towards lung bases.	Early broadening at right lung base.	Not taken.	17. 3.49	22. 5.50: "Extremely well. No cough and never gets tired now. Doing well at school."
8	M.S.	F.	8	4 years.	Catarrhal changes at both lung bases.	Suggestive of early bronchiectasis, left cardio-phrenic angle.	No evidence of bronchiectasis.	16. 6.50	31. 5.50: "Has been extremely well. No cough."
9	C.O'B.	M.	12	11 years.	Some bronchitic changes, especially at lung bases.	Suggestive of slight broadening at left lung base.	Not taken.	10. 6.48	18. 4.50: "Has been very well. Has no cough at all now."
10	C.G.	F.	6	5½ years.	Increased basal markings suggest bronchiectasis.	Some dilatation at right lung base.	No evidence of bronchiectasis.	16. 2.50	4.11.50: "Very well indeed. No cough now and no asthma."
11	E.S.	F.	6	10 weeks.	Chest clear.	Posterior basal bronchi show suspicious broadening.	No evidence of bronchiectasis.	12. 1.50	17. 1.51: "Has been very well. No cough. Third in class. No colds. Seems better in every way."
12	G.T.	F.	4	2 years.	Possible early bronchiectasis at left lung base.	Blocking of terminal bronchi at both lung bases.	No evidence of bronchiectasis.	14.12.50	14.12.50: "Appears greatly improved. No cough."
13	G.O'C.	M.	6	5½ years.	Chest clear.	Left lung base shows suspicious dilatation.	No evidence of bronchiectasis.	5. 1.50	10. 7.50: "Has been very well. Does not cough now."
14	D.L.	M.	6	4½ years.	Chronic bronchitic changes.	Early dilatation at both lung bases.	No evidence of bronchiectasis.	23. 2.50	6. 3.50: "Extremely well. Very pleased with him. Going to Scotland."
15	W.R.	F.	7	4 years.	Pronounced bronchitic changes.	Right side shows small area of bronchiectasis and atelectasis.	No evidence of bronchiectasis.	23. 3.50	24. 8.50: "Is 100%. No cough. Eats and sleeps well. Top of class. Teacher says she is absolutely a new child."
16	J.A.	F.	5	6 months.	Bronchitic changes in both lungs, especially at bases.	Minimal saccular dilatation at left cardio-phrenic angle.	No evidence of bronchiectasis.	5. 1.50	18.10.50: "Extremely well. No cough at all. Is greatly improved in every way."
17	M.S.	F.	5	4½ years.	Mild catarrhal changes in both lungs.	Early bronchiectasis at right cardio-phrenic angle, and atelectasis.	No evidence of bronchiectasis.	24. 8.50	1.11.50: "First winter since eighteen months old she has not been in bed. We can hardly realize she is the same child, she is so different. Did not like school. Now loves it."
18	K.W.	M.	5	10 months.	Strong suggestion of bronchiectasis at lung bases.	Suggestive of bronchiectasis at left cardio-phrenic angle.	No evidence of bronchiectasis.	20. 7.50	3.11.50: "Very well. No cough. Seems to be nothing wrong with him. Is very different."
19	P.McC.	F.	5	2 years.	Lung fields appear clear.	Suspicious area at left cardio-phrenic angle.	No evidence of bronchiectasis.	26.10.50	9.10.50: "Very well indeed now. No cough. Is very much brighter."
20	T.B.	M.	5	2 years.	Increase in bronchial markings at right lung base.	Suspicious appearances in left basal bronchi.	No evidence of bronchiectasis.	15. 2.51	15. 2.51: "Very slight cough indeed. Playing much better. Has altered considerably. Can take him out now. Would not before because cough was so bad."

Hæmoptysis.

Three patients in each group had had hæmoptysis, which in all but one case was only slight. The one with the more severe bleeding was in Group II.

Allergy.

In Group I 13 patients had allergic manifestations, while the parents of five others were allergic—a total of 18 with an allergic background. In Group II 12 had allergic manifestations, while the parents of three others were allergic —a total for this group of 15 with an allergic background. Furthermore, in this group there were one orphan and two adopted children with no available family history.

It will be observed, then, that a very high proportion of pre-bronchiectatic children have an allergic background, and it is highest in those with early signs of bronchiectasis.

Colds.

In Group I 23 patients, and in Group II 14 patients, stated that colds were very common before treatment, but that they were uncommon or even rare afterwards. In only one case (Group II) did the patient complain of frequent colds after treatment, but these were all in the head.

Upper Respiratory Tract Infection.

In Group I 18 patients had some upper respiratory tract infection with slight nasal discharge or definite sinusitis, mostly antritis. In two cases double intranasal antrostomy was performed, while in the remainder "Argyrol" drops (10%) were used.

In Group II 11 patients had some upper respiratory tract infection. In this group five underwent a double intranasal antrostomy and the remainder were treated with "Argyrol" drops. The end result in all cases in both groups was a clear upper respiratory tract, except in one case in which a slight amount of discharge was still present. These figures are lower than those found in established bronchiectasis.

Weights.

These children often look quite well and their weights are inclined to be higher than normal in comparison with the figures given by Clements and Machin for infants and school children of New South Wales in 1937. When their figures for normal children are considered, the weight chart shows that a large percentage of patients are overweight in both groups, as well as patients with established bronchiectasis in the same age group. It will also be noticed in these groups that the average excess weight over the normal is rather high in children who are over-weight, while in those who are below normal weight the difference is not so pronounced.

It has been suggested that this increase may be due to the fact that these children are not so active as the normal child. This may explain many cases, but some children show no apparent diminution in their activities. Also, if this was the sole factor, we should not expect to see the immediate increase of weight we usually find under treatment; rather might we find a preliminary fall as the patients become more active. The details of weight are set out in the following tabulation:

Group I:

Eighteen patients were overweight (average, 9¼ pounds).

Two patients were of normal weight.

Ten patients were below normal weight (average, 5¾ pounds).

Group II:

Seventeen patients were overweight (average, 11 pounds).

Three patients were below normal weight (average, 2½ pounds).

Seventy-Three Established Bronchiectatics of the Same Age Group:

Forty-three were overweight (average, 9½ pounds).

Seven were of normal weight.

Twenty-three were below normal weight (average, six pounds).

All these weights were recorded at the commencement of treatment.

Treatment.

The main treatment in all these cases was bronchoscopic aspiration and lavage. In Group I the average number of bronchoscopic treatments was five, spread over an average period of five months, while in Group II the average number of treatments was 13, spread over an average period of fifteen months.

Bronchoscopic Findings.—In Group I the amount of mucopus washed out varied from "slight" to "moderate". At the end of treatment in only two cases was a very slight trace of mucopus still present. In Group II the amount varied to the same degree, and in this group also two patients still showed a slight trace of mucopus at the end of treatment. In all other cases in both groups the lavage fluid was returned clear or containing just a shred or two of mucopus present. In both groups congestion was found at times, but it was more common in Group II, in which it was likely to be found in the region of the suspected bronchi, around the orifices of which granulations were occasionally found.

Treatment, Other than Bronchoscopic.—Treatment before consultation had usually been with all types of cough mixtures, varying drops in the nose, trips to the country, and in the later cases the administration of sulphonamide drugs and penicillin.

Throughout both groups, all but five patients had had tonsillectomy performed as treatment before coming up for consultation. This operation was usually performed around the age of four years, but after operation the patients showed no pronounced or only very temporary improvement. Of the five patients who had not been operated on, I removed the tonsils and adenoids from one during treatment as they were hypertrophied and unhealthy. This was the patient who had reported for treatment after ten weeks.

In this series of cases, treatment given besides bronchoscopic lavage in Group I was as follows: inhalations in about one-third of the cases; usually the administration of "Adexolin"; in some, breathing exercises and sometimes towards the end of treatment a trip to the country. In Group II similar treatment was given, but postural drainage and more frequent breathing exercises were usually added. In both groups iron preparations were given when indicated, and the diet was checked and perhaps altered or added to. An autogenous vaccine was given in two cases, one in each group.

Summary of Findings.

In Group I (Table III) the findings on a plain X-ray examination usually consisted of an increase in the basal markings. In two cases no changes were present, though the patients had typical histories and bronchoscopic findings. In all cases in this group the bronchographic appearance was normal. Also tabulated is a recent comment by the parents on the effect of treatment and the present condition of the child. Follow-up plain X-ray films have not been taken as a routine measure, but when they were taken the greater proportion have been free from abnormality.

In Group II (Table IV) it will be seen that the first bronchograms all showed areas arousing suspicion of dilatation or atelectasis, while those taken at the end of treatment revealed no abnormality in the lungs. Here also comments of the parents or guardians are given.

Comment.

In this paper I have tried to point out the absolute necessity of giving these little patients the advantage of early bronchoscopic drainage, without which it would appear that they may eventually develop irreversible bronchiectasis. If they are not caught early, there would appear to be still

quite a reasonable chance in some cases of preventing the development of a permanent lesion. The outstanding finding is how long some of these children can go on with severe symptoms and still return to normal under bronchoscopic treatment. It will also be seen that these patients require the help of bronchoscopic lavage and aspiration over and above all other forms of treatment before they give any response.

Most of these children are cooperative under local anæsthesia, and all the patients in this series have been treated with a local anæsthetic except when a bronchogram was taken, some then having a general anæsthetic to prevent any movement.

For estimating the end results of treatment I would draw your attention to the findings by bronchoscopic lavage before and after treatment, the later bronchograms in Group II, and the final clinical findings and the parents' comments in both groups.

Surely if the problem of pre-bronchiectasis was tackled thoroughly we would find that the number of cases of irreversible bronchiectasis was diminished considerably and a good deal of ill health, loss of work and major surgical treatment would be eliminated.

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A CHLORIDE-TESTING PAPER FOR CLINICAL USE BY UNTRAINED PERSONS.

By JOHN DEVINE.

From the Royal Melbourne Hospital, Melbourne.

THE test paper here described is intended for use by nurses or untrained persons in the rapid clinical estimation of the chlorides in the urine or other body fluids.

The method was originally developed to enable nurses to test the chloride concentration of all specimens of urine passed by patients who were having intravenous administration of fluids. It has been found to be applicable to other body fluids, including blood, and there are also commercial applications.

The method of Fantus (1936) for the quantitative estimation of the chlorides in urine requires more skill than is usually allowed for in the average nursing staff, and is too tedious to be used for the examination of every specimen of urine passed by a patient. Two reagent bottles, a dropper and test tubes are used, and the test tube and dropper must be kept clean between estimations.

When the paper-indicator method as herein reported is employed, the estimation of the chlorides in urine is made by a nurse or other untrained person, who tears a small piece of indicator paper out of a book (similar to a litmus book), and who, having taken care that the paper becomes wet immediately, notes the time taken for the indicator paper to change from brown to white. The strength of the first paper commercially available has been provided for testing the urine of patients who are undergoing intravenous therapy, and is so adjusted that a change occurs in five to six seconds if the urinary chloride content is 0·3%. Therefore, if the change in colour of the paper occurs immediately or within five or six seconds, the patient has over 0·3% of sodium chloride in the specimen of urine being tested.

Since there is a time difference of forty-three seconds between the colour change with 0·3% and 0·1% sodium chloride solution, it follows that appreciable errors in timing by the nurse cause but slight errors in the results.

Urinary chloride estimations are of value in keeping a patient in "chloride balance" if possible anomalies are remembered when the results are interpreted.

Blalock (1940), quoting Paine (1940), makes the following statement:

> Chlorides are threshold substances and are excreted by the kidneys—only after the body has retained all it requires for metabolic needs. Therefore, one can be reasonably certain that a patient who is fully hydrated and excretes 3 grams or more of chloride expressed as sodium chloride in his urine daily, is in adequate sodium chloride balance.

Wangensteen (1945), while discussing the post-operative management of intestinal obstructions, states:

> Once the losses of sodium chloride sustained in vomiting have been replenished, unless the gastro-duodenal aspirations are great, 4 to 9 grams of sodium chloride will meet the situation adequately. If more than 3 grams are excreted daily in the urine, even this amount (9 grams) is too much.

Mason and Zintel (1946) make the following statement:

> Daily estimation of the plasma chlorides would prove expensive and time-consuming. The salt content of the urine may be followed daily. As long as the patient excretes more than 1 gram of salt per day in the urine, there is little danger of serious salt depletion. (Bartlett) . . . if excesses of salt are being given, large amounts of salt are usually found in the urine.

Scribner, Power and Rynearson (1950) attach importance to a daily estimation of the water-chloride balance, in which they save and measure the volume and chloride concentration of all drainage and excreta, and add an allowance for fluid and chloride lost in the sweat.

> The application of chloride balance technics to the clinical management of problems of fluid balance has not been practicable in the past because of the expense and technical difficulties of obtaining chemical determinations. However, a new method for the rapid, accurate determination of chloride at the bedside makes the measurement of chloride balance a practical clinical procedure. The method is based on the direct titration of the chloride with mercuric nitrate. . . . Reagents are stored in small bottles which are taped together to form a portable test set. Solutions are measured with tuberculin syringes which fit into the caps of the bottles . . . a bottle of standard salt solution is included in the test set.

Such a method is scarcely simple, and their article shows a photograph of a "fluid balance technician", who pushes a laden hospital cart to visit patients daily, and keeps charts, and estimates the pH and chloride contents of the various excreta and output from the patient for the preceding twenty-four hours. She also analyses and estimates the oral and parenteral fluid and salt intake during the same period of time.

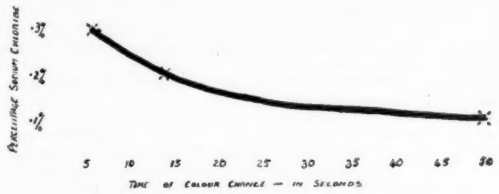

FIGURE I.

Graph showing the relation between the time of colour change and the percentage w/v of sodium chloride in aqueous solutions. (Paper supplied by Felton, Grimwade and Duerdins, Proprietary, Limited, as "Chlorest Paper A".)

While such a water-chloride balance estimation avoids relying too much on the often-misleading plasma electrolyte estimations and on the clinical signs and symptoms of electrolyte imbalance (which do not occur until fluctuations from normal are of considerable magnitude), the whole procedure is cumbersome and requires a skilled technician.

With the simple indicator-paper method of chloride concentration estimation herein described, it has been found simple and quick for the nursing staff to estimate the chloride concentration of all excreta and body fluids removed, and so to allow for the planned replacement of the same amounts of chlorides in fluids given.

Chloride-testing Paper in Use.

Clinical trials of the indicator-paper method herein reported included two months' continuous use of the indicators by Sister G. R. Anderson and her trained and trainee nursing staff in Ward N2 of the Royal Melbourne Hospital. These and other trials (over a period of eighteen months) showed that it was necessary to protect the test papers from prolonged exposure to very strong daylight (by opaque covers to the indicator "books") and from contamination by the often-wet hands of the nursing staff (by waterproof covers).

This paper method has since been applied to estimation of the chlorides in blood, serum, plasma and cerebro-spinal fluid. It has been employed in trials by the patients themselves for testing the concentration of chloride in their own urine; when a low chloride intake had been prescribed (as for hypertension), the patients were able to test each specimen of their own urine, and to modify their diet according to the chloride concentration found.

A strength of paper particularly designed for use in the estimation of chlorides in the urine of patients undergoing intravenous therapy has been developed on a commercial basis by Felton, Grimwade and Duerdins, Proprietary, Limited (in particular by Mr. E. I. Rosenblum and Mr. W. S. Taylor), under the name of "Chlorest Paper A". This paper has a clear-cut endpoint at which its brown colour turns to white when it is dropped into the urine. The following figures for the time of colour change were noted:[1]

- 0·3% sodium chloride in the solution changes the paper in six seconds.
- 0·2% sodium chloride in the solution changes the paper in thirteen to fifteen seconds.
- 0·1% sodium chloride in the solution changes the paper in forty-eight seconds.

Thus if the colour change occurred within six seconds the urinary chloride excretion was considered to be adequate.

While this particular paper was standardized at 20° C., it has been found that temperature variations within the usual range caused no clinically significant difference in the results. It was found unnecessary to wait until the urine cooled before testing it.[2]

Summary.

An indicator-paper is reported which was designed primarily for the rapid and repeated estimation by nursing staff, patients or untrained and inexperienced persons of the chloride concentration of individual or of all the specimens of urine passed, and with a sufficient degree of accuracy for clinical purposes. The chloride concentration is estimated by tearing off a small piece of indicator-paper and dropping it into the urine or solution being tested and noting the time taken for the colour to change from brown to white.

Acknowledgements.

Many helped, but I would like to express my thanks to the following: Professor R. D. Wright; Mr. Russell Grimwade, Mr. E. I. Rosenblum and Mr. W. S. Taylor, of Drug Houses of Australia. To Miss Beryl Splatt, clinical biochemist at the Royal Melbourne Hospital, I should particularly like to extend my thanks.

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Appendix.

The time of colour change of the indicator-paper was obviously dependent on a number of factors, and the following theoretical considerations were studied before the first home-made papers were tried.

The rate of the reaction should be predictable, since the law of "mass action" was applicable.

For a reaction to occur between the solution of chlorides in the urine or fluid being examined and the chemical reagent (water-soluble silver chromate sparingly precipitated in the paper during manufacture), the reagent has to be in solution. Since the smallest molecules travel fastest in capillary networks, such as paper, water molecules in solution would travel faster than chloride ions and act as a solvent for the reagent (for example, paper chromatography).

The time taken for the reagents in the paper to be dissolved would therefore depend on the character, capillarity, density, thickness and size of the test papers; on the

[1] The table of times of colour change for "Chlorest Paper A" with various strengths of sodium chloride solution in water are given by the makers as follows: 0·5% w/v changes colour in 1 to 1·5 seconds; 0·4% w/v changes colour in 2 to 3 seconds; 0·3% w/v changes colour in 5 to 6 seconds; 0·2% w/v changes colour in 13 to 15 seconds; 0·1% w/v changes colour in 45 to 55 seconds.

[2] The effect of different temperatures is as follows: 20° C., seven seconds for 0·3%; 30° C., six seconds for 0·3%; 20° C., thirty seconds for 0·15%; 30° C., twenty-five seconds for 0·15%.

humidity and therefore the amount of pre-solution; on the temperature of the solution being tested; on the presence in the solution being tested of substances (for example, protein) which might react with the indicator reagents to form a barrier to "wetting"; and on many other factors. Most of the factors produce negligible variations in results, and are of no importance to the results as required for clinical use, and most are nullified by manufacture of the papers in tested and standardized "batches".

The amount of indicator reagent and therefore the size of the test piece of paper must be kept very small in comparison with the volume of fluid being tested, and so that alterations in the concentration of chloride in the fluid being tested brought about by "mass action" with the reagent in the paper would be so small that its chloride concentration might be considered as a constant. This modifies and simplifies the graph of the rate of the reaction.

If the reagents, paper and temperature were to be kept constant, the time-rate of reaction should bear a relation to the concentration of the solution being tested, and would so serve to measure such a concentration.

Papers, applied alternately, were therefore made by impregnation with silver nitrate and potassium bichromate, and it was found that the above postulate held, and that the time of reaction was proportional to the chloride concentration in the solution being tested. The test reagent was thus sparingly water-soluble silver chromate impregnated in the meshes of the filter papers during manufacture.

It follows that care must be taken that the papers are immediately and quickly "wet" by the solution being tested—that is, that they do not rest on the surface held by surface tension, being in contact with only a small amount of the solution being tested.

Reviews.

SURGERY.

"PRINCIPLES AND PRACTICE OF SURGERY", by J. K. Berman,[1] is a sincere attempt to present the ever-expanding subject of surgery in such a way that when the student passes from his study of the exact sciences to the inexact subject of surgery, the change will occur with as little of a "clash of changing gears" as possible, and in a manner so that his earlier studies are of some use to him.

Ever since Isaac Newton published his *"Principia Mathematica"* in 1687 and formulated the law of gravitation, scientists have been busily searching the universe for further evidence of its reason and order. It is therefore not to be wondered that someone should endeavour to reduce the untidy, sprawling and quasi-scientific subject of surgery to a reasonable, reasoned and ordered state, so that the student would not have to forget all his early training when he commences to study surgery—a mixture of science and art. Though it is reasonable to assume, from the continued trend which began at least as early as in the times of Imhotep, who lived in Egypt three centuries before the birth of Christ, that further study will make surgery still more of a science and less of an art, at its present stage of development there must be difficulties in fitting the part which remains "art" into a treatise which is intended to be logical and scientific. Similar difficulties were experienced by Newton in his *Principia* in the 17th century. It may well be that many more years will elapse before the subject of surgery can be treated in an entirely logical and scientific manner.

That Berman's book is incomplete and at times not in accord with the latest scientific thought is but a minor defect—it will be of considerable help to undergraduates as an introduction to their final and their hospital years. There would be few who would make any criticisms if the book was given, after the manner of C. H. Best and N. B. Taylor, some such title as "The Physiological and Pathological Basis of Surgical Practice".

The first part of the book is general, and deals with pathology and general and local reactions to injury, and is outstanding even in such an excellent book. The second part of the book deals with the diseases and injuries of the various special systems. This very intricate work features a fairly comprehensive bibliography at the end of each section.

The reader's attention is first caught by a short and interesting survey of the history of medicine. Then comes a description of the response of the body to local injury. Mention is made of factors which may result in non-healing of a wound.

The author refers often to underlying physiological facts; for example, that skin grafts "take" because of the activity of the prickle cell layer of cells in the skin, which move by pseudopodial prolongation, and that the *stratum germinativum* is even more important in the process of this "taking".

While discussing ulcers and gangrene of the legs and tests for peripheral circulation, the author mentions arteriography as being not without danger. Oscillometry, a venous return test, and changes in skin temperature are also used in the estimation of the peripheral circulation.

Liberal and excellent illustrations are a feature, and none are more useful than the black and white illustrations which serve to illustrate techniques or procedures which have been so grouped that they will be easy to memorize.

In the general section, the reader is continually encountering useful data which are unavailable elsewhere, or at least not without a considerable search. Under the section on thermal injury, diagrams and tables are given which make possible the accurate estimation of the percentage of the body area which has been burnt.

The section devoted to acid-base balance is so sensible and logical and helpful that it alone would make the whole book well worth while.

Shock may be "neurogenic" or primary; it may also be hæmatogenic or secondary. Among the other types of shock mentioned are "cardiogenic" shock; "toxæmic" shock; "thoracic" shock; and shock due to "crush syndrome" or ischæmic muscle necrosis, traumatic œdema or traumatic anuria; blast concussion, acceleration shock, and particular types of shock due to extremes of environmental temperature.

While there is much to recommend the general portion of the book, a rational and logical approach to the surgery of particular systems and regions has not resulted in such a happy arrangement or classification as has been achieved in the discussion on general surgical conditions. Despite this, candidates for higher degrees will find clear pages of illustrations of the various methods, such as those of dealing with aneurysms (many of them historical only), which will be of great use in their study for higher examinations. Photographs are used to demonstrate clinical conditions and specimens, and wherever possible anomalies are explained by a pictorial review of the embryology.

This book will be of very great help to undergraduates, who will use it as an introduction to surgery and to their hospital years. Post-graduates will use its up-to-the-minute discussion of a number of subjects in their study for higher degrees.

[1] "Principles and Practice of Surgery", by J. K. Berman, A.B., M.D., F.A.C.S.; 1950. St. Louis: The C. V. Mosby Company. Melbourne: W. Ramsay (Surgical) Proprietary, Limited. 9½" × 6½", pp. 1396, with 429 illustrations. Price: £7 17s. 6d.

Books Received.

[The mention of a book in this column does not imply that no review will appear in a subsequent issue.]

"Surgical Nursing and After-Treatment: A Handbook for Nurses and Others", by H. C. Rutherford Darling, M.D., M.S. (London), F.R.C.S. (England), F.R.F.P.S. (Glasgow), and T. Edward Wilson, M.D., M.S., M.Sc. (Melbourne), F.R.C.S. (England), F.R.C.S. (Edinburgh), F.A.C.S., F.R.A.C.S., M.R.A.C.P.; Tenth Edition; 1951. London: J. and A. Churchill, Limited. 7¼" × 5", pp. 640, with 188 illustrations. Price: 16s.

The ninth edition was published in 1946 and reprinted in 1947.

"Hospital Staff and Office Manual", by T. M. Larkowski, M.D., F.A.C.S., and A. R. Rosanova, R.Ph., M.D.; 1951. Great Neck, New York: Romaine Pierson Publishers, Incorporated. 7½" × 5", pp. 442, with many illustrations. Price: $4.95.

The book is claimed to provide a ready and practical review of all phases of medical practice.

"Good Health with Diabetes: A Patient's Handbook", by Ian Murray, M.D., F.R.F.P.S.G., F.R.C.P.E., and Margaret B. Muir, S.R.N.; Second Edition; 1951. Edinburgh: E. and S. Livingstone, Limited. 7½" × 5", pp. 48. Price: 2s.

The object of this booklet is to enable the diabetic "to educate himself so that his dietary may be satisfying, and varied according to choice, but of the same daily carbohydrate value".

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All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given without abbreviation: surname of author, initials of author, year, full title of article, name of journal without abbreviation, volume, number of first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for reproduction are invited to seek the advice of the Editor.

MATERNAL CARE AND MENTAL HEALTH.

THOSE who rear children know that "as the old cock crows the young cock learns". They also realize that the earlier the training of a child is commenced, the more effective and lasting is the result likely to be. In the Book of Proverbs we read: "Train up a child in the way he should go: and when he is old, he will not depart from it.". It is the mother of a child who makes most impression on the young mind and lays the foundations of the building of character. Napoleon is recorded as having said that the future destiny of the child was always the work of the mother, and Abraham Lincoln declared that all that he was or hoped to be he owed to his "angel mother". The altered attitude of present-day parents, and of mothers in particular, to their offspring, and in many cases the consequent neglect of them, have had a profound influence on present-day children. Again, many mothers who, with the best will in the world, have tried to care for their children, are handicapped either by their ignorance or by altered conditions which are anything but helpful, even to understanding and careful mothers. The recent war has, of course, made the lives of many children nothing but a complete hardship. The difficulties have been so great that at the third session of the Social Commission of the United Nations, held in April, 1948, it was decided to make a study of the needs of homeless children. These homeless children were described as those "who are orphaned or separated from their families for other reasons and need care in the foster homes, institutions or other types of group care". The study was to be confined to "children who were homeless in their native country" and refugees from war or other disaster were explicitly excluded. The result was that the World Health Organization arranged to have an investigation carried out by Dr. John Bowlby, the director of the Child Guidance Department of the Tavistock Clinic, London, and consultant in mental health to the World Health Organization. Bowlby's report has been published in a special number of the *Bulletin of the World Health Organization*. During his investigations Bowlby visited France, the Netherlands, Sweden, Switzerland, the United Kingdom and the United States of America. He has divided his report into two parts. The first deals with the adverse effects of maternal deprivation and the second with its prevention.

Bowlby points out that it is the mother, rather than the father, with whom the young child is chiefly concerned. It is the mother who feeds and cleans him, keeps him warm and comforts him. He turns to the mother when he is in distress. In the eyes of the young child the father plays second fiddle and his value increases only as the child's vulnerability to deprivation decreases. Fathers provide for their wives and enable them to devote themselves unrestrictedly to the care of the infant and toddler; by providing love and companionship they support her emotionally and help her to maintain "that harmonious, contented mood in the aura of which the infant thrives". Bowlby therefore refers constantly to the mother-child relation and has little to say about the father-child relation; the value of the father as the economic and emotional support of the mother is assumed. In reviewing the evidence on the effect of deprivation, Bowlby points out that it falls into three main classes. The first class comprises evidence from studies by direct observation of the mental health and development of children in institutions, hospitals and foster homes. The second class comes from studies which investigate the early histories of adolescents or adults who have developed psychological illness; the third from studies which follow up groups of children who have suffered deprivation in their early years, with a view to determining their state of mental health. The direct studies are the most numerous, and Bowlby points out that they make it plain that, when the child is deprived of maternal care, his development is almost always retarded—physically, intellectually, and socially—and that symptoms of physical and mental illness may appear. All children under about seven years of age seem to be vulnerable, and some of the effects are clearly discernible within the first few weeks of life. Bowlby quotes from an article by H. Bakwin[1] the following summary of his observations:

> Infants under six months of age who have been in an institution for some time present a well-defined picture. The outstanding features are listlessness, emaciation and pallor, relative immobility, quietness, unresponsiveness to stimuli like a smile or a coo, indifferent appetite, failure to gain weight properly despite the ingestion of diets which, in the home, are entirely adequate, frequent stools, poor sleep, an appearance of unhappiness, proneness to febrile episodes, absence of sucking habits.

He also gives a reference to another article by Bakwin[2] in which he reports that at the Bellevue Hospital, New York, nurses and internes are encouraged to manipulate the infants at every opportunity and parents are invited to visit them. These measures have not been found to affect the health of the infant adversely. Bowlby quotes several groups of authors who confirm Bakwin's findings, and he adds that this diverse evidence from reputable workers leaves no room for doubt that the development of infants in institutions deviates from the norm at a very early age. He refers also to studies showing similar retardation in the second and later years. W. Goldfarb

[1] *Journal of Pediatrics*, Volume XXXV, 1949, page 512.
[2] *American Journal of Diseases of Children*, Volume LXIII, 1942, page 30.

made a study of 30 children aged from thirty-four to thirty-five months. From the age of four months half of these children had lived in institutions and the other half in foster homes. When the intelligence quotient of the children was estimated, it was found that the children in the foster homes had a quotient which averaged 96, while those in an institution had an average quotient of 68. Bowlby points out that in the studies to which he refers care has been taken by most of the investigators to ensure that the controlled groups, brought up either in their own homes or in foster homes, are of a similar social class and as nearly as possible spring from similar stock. The findings cannot therefore be questioned on the ground that children in institutions are essentially different from those with whom comparisons have been made. As an indication of how far-reaching the effects of maternal deprivation may be, the work of Binning, mentioned by Bowlby, may be quoted. Binning concluded after studying 800 Canadian school children, that changes in the speed of growth were frequently emotional in origin and might take the form of either an acceleration or a lag. He wrote:

> We found that events in the child's life that caused separation from one or both parents—death, divorce, enlistment of a parent—and a mental environment which gave the child a feeling that normal love and affection was lacking, did far more to damage growth than did disease, was more serious than all other factors combined in this day of full employment and family allowances.

These considerations are possibly incidental to the present subject, but Bowlby points out that, if confirmed, these results will open up new possibilities of research into the interrelation of psyche and soma, and will provide the clinician with a simple and reliable tool. He emphasizes that Binning's conclusions must for the present be regarded as tentative. In regard to retrospective studies, those which combine the second of the three main classes of evidence, interesting findings are presented. Bowlby points out that in the late 1930's at least six independent workers were struck by the frequency with which children who committed numerous delinquencies, who seemed to have no feelings for anyone, and who were very difficult to treat, were found to have had grossly disturbed relationships with their mothers in their early years. "With monotonous regularity each puts his finger on the child's inability to make relationships as being the central feature from which all other disturbances sprang, and on the history of institutionalization or . . . of the child's being shifted about from one foster mother to another as being its cause." It may be remembered that Hervey Cleckley in his book "The Mask of Sanity", discussed in these columns on January 27, 1951, reached much the same sort of conclusion in regard to adult "psychopaths". Similar conclusions are reached in the follow-up studies, the third of the three main classes providing evidence for this investigation. Bowlby refers to the work of several authors. One investigation by Goldfarb is quoted in which 15 pairs of children were investigated when they were at ages ranging from ten to fourteen years. One set had been in an institution from the age of six months until they were three and a half years old; the other set had not had this experience. Conditions in the institution conformed to the highest standards of physical hygiene, but lacked the elementary essentials of mental hygiene. Babies below the age of nine months were each kept in their own little cubicles to prevent the spread of epidemic infection. Their only contacts with adults occurred during the hurried moments when they were dressed, changed, or fed by nurses. The findings in the two groups, which are set out in a table, confirm Bowlby's general contention. Bowlby quotes one observer who concluded that constitutional factors were at least as important as environmental factors in social maturation, but owing to the shortcomings which he finds in this work, he cannot accept it as calling in question the almost unanimous findings to which he himself has referred. He concludes that there is no evidence which could be held to invalidate his hypothesis. Incidentally, he remarks that the conditions provide unusually rich opportunities for research in child development.

The question will naturally be asked what is to be done for a child who is known to have been deprived of the early maternal influences which should act upon him. Whether anything much can be done will depend on the stage which he has reached. Bowlby describes certain phases in the development of a child's capacity for human relationship. The first phase is that during which the infant is establishing a relation with a clearly identified person—his mother; this is normally achieved by five or six months of age. The second phase is that during which the child needs an ever-present companion; this usually continues until about his third birthday. The third phase is that during which he is becoming able to maintain a relationship with his mother *in absentia*. During the fourth and fifth years such a relationship can be maintained only in favourable circumstances and for a few days or weeks at a time; after seven or eight years of age the relationship can be maintained, though not without strain, for periods of a year or more. Bowlby points out that if the first phase of development is not completed during the first twelve months or so, there is the greatest difficulty in making it good because the character of the psychic tissues has become fixed.

With these admittedly superficial observations on this aspect of the subject, attention must be directed to the second half of Bowlby's document, that dealing with the prevention of maternal deprivation. In general terms the prevention of maternal deprivation may be summed up in the words, the conservation of family life. This is a subject on which many articles might be written; and no doubt time and time again in past ages the break-up of family life has been deplored. Fortunately the maternal instinct is so ingrained in woman that large numbers of children are still cherished and cared for even in the present hurrying and workaday world. It is unfortunately true that many women who wish to devote proper care to their children are unable to do so because they are compelled to go to work and earn their living. It is also true, as already stated, that many women, able and willing to care for their children, have no idea of how they should go about doing it. The first problem therefore to be faced is economic and the second is educational. It is unlikely that either will be solved, but that does not mean that we should sit still and not attempt a solution. To pluck one brand from the burning, or in other words, to allow one child to lead a full life, is something worth while. Bowlby discusses the causes of family failure in western communities and makes special reference to psychiatric factors. He mentions such subjects as

illegitimacy, chronic illness or incapacity of a parent, instability or psychopathy of a parent, social calamities such as war and famine, death of a parent, desertion by one or both parents, separation or divorce, and so on. It is the tradition in western communities for near relatives to care for children when the natural home group for any reason has failed, and no account of the causes of the homelessness of a particular child is complete unless the reason for the failure of relatives to act is given. Bowlby has a good deal to say about adoption, and names three principles in regard to a baby's suitability for adoption. The first is that an assessment of the child's genetic potentialities requires the opinion of a person with training in human genetics and that in no case should an adverse decision be reached without the opinion of a competent person. The second principle is that psychologists should be thoroughly familiar with the predictive value of their tests and with the effect of deprivation, illness and other environmental factors on test performance. The third is that even if a child's state or prognosis about his future is not wholly favourable, an attempt should still be made to see whether there may be adoptive parents who, after being given full knowledge of the facts, are prepared in a realistic mood to accept him. Group care, or the disposal of children in residential nurseries and so on, is also mentioned. In all these circumstances any success that is achieved will be the result of some personal link forged between the child and the person who is in charge of him. It is interesting to note that Bowlby declares that group residential care is always to be avoided for those under about six years, but that it is suitable for short-stay children between six and twelve years of age, and both for short-stay and some long-stay adolescents. Group care cannot be avoided for some maladjusted and sick children. In his concluding remarks Bowlby states that no country has so far tackled seriously the problem of children deprived of a normal home life. Thus the World Health Organization has put every country in its debt by this study of Bowlby's. It is to be hoped that wide publicity will be given to it—there is still "a lack of conviction on the part of governments, social agencies, and the public that mother-love in infancy and childhood is as important for mental health as are vitamins and proteins for physical health".

Current Comment.

WHOOPING-COUGH AND PREVENTIVE VACCINATION.

ALTHOUGH most of our readers will have the opportunity of examining it for themselves, the recently published report of a Medical Research Council investigation into the prevention of whooping-cough by vaccination[1] is sufficiently important to demand special comment. Confusion has unfortunately arisen in the past from conflicting reports by investigators of standing on the prophylactic value of pertussis vaccines. Notably, the unfavourable findings of the Whooping-Cough Immunization Committee of the Medical Research Council, based on controlled trials in England in 1942-1944, contrasted with favourable results reported from elsewhere, particularly from North America. Concluding that the difference in the results must be related to the vaccines used, the Whooping-Cough Immunization Committee decided to carry out a new series of field trials with vaccines prepared in different laboratories, including some of American origin which had been reported to give substantial protection. The present report shows that this approach was amply justified. Five batches of vaccine were used—three from the United States, two from England—and an extensive well-controlled field trial was made with 1500 children between the ages of six and eighteen months. Each vaccine gave substantial protection. With Sauer vaccine prepared by Parke, Davis and Company, of Detroit, the ratio of the attack rate per 1000 child-months of observation in the vaccinated to that in the unvaccinated group was 1:2·5. There was a slightly greater degree of protection with each of two vaccines prepared by Glaxo Laboratories, Limited, in England, the corresponding ratios being 1:3·3 and 1:3·6. Two vaccines obtained from the Michigan Department of Health gave a considerably greater degree of protection than the others; the ratios with these were 1:7·3 and 1:10·8. The general observation for all vaccines was that cases of pertussis that occurred in the vaccinated group were on the average less severe and of shorter duration than those in the unvaccinated group. During the periods of observation (two to three years) there was no evidence of a waning in the degree of protection afforded by the pertussis vaccines.

These findings, coming as they do from a committee that previously was not favourably impressed, are reassuring. The work of McCloskey and others, which at least suggests a relationship between pertussis vaccination and the incidence and localization of paralysis in poliomyelitis, has cast pertussis vaccination into something of a shadow. The reaction of many informed pædiatricians has been that the risk involved, however real it may be, does not warrant the outlawing of vaccination against such a lethal disease as whooping-cough, provided that vaccination is effective. The great service done by the present report is in clearing the existing confusion. It makes it clear that pertussis vaccines may vary considerably in their protective capacity. This variation in potency, the report states, is possibly related to the many variables concerned in the preparation of vaccines. Much work yet needs to be done in the investigation and comparison of methods of preparation, and a satisfactory laboratory means of assaying prophylactic activity has yet to be found. Investigations on these matters are under way, and no doubt we shall have to be patient in awaiting conclusions. The important point, however, is that we may look with renewed confidence to the control of whooping-cough by vaccination.

[1] *British Medical Journal*, June 30, 1951.

FAINTING IN TOILS.

THE Mock Turtle's school curriculum included Fainting in Coils, so he told Alice, but this must have been a dull subject compared with a nineteen-year-old student's experience of fainting in toils described in a recent report by A. D. M. Greenfield.[1] Greenfield, who is Dunville Professor of Physiology in Queen's University, Belfast, does not use the actual phrase of our heading, but it seems to fit his account of a fainting student who fell into the investigational toils of an enthusiastic physiologist. It seems that the student fainted and fell while watching blood being drawn from another person's arm vein. He had previously been healthy (and has remained so since), so an attempt was made to cause him to faint in the same way under close observation, in order that the responses of an emotional faint could be compared with those of post-hæmorrhagic fainting. This time, however, though the spirit was willing, the flesh was strong, and fainting needed much encouragement. While preparations for venesection (on another person) were being made, as obviously as possible, the student's forearm blood-flow and arterial pressure were observed to rise from their resting values and the heart rate increased. No further increases

[1] *The Lancet*, June 16, 1951.

occurred when blood was withdrawn, and rather inconsiderately he failed to faint. A needle was then inserted into the student's arm, though no blood was withdrawn; the forearm blood-flow again increased, but no faint occurred. It was therefore decided (by Count Dracula, presumably, though Professor Greenfield does not mention him by name) to increase the stimulus by asking the student to drink the few millilitres of blood he had seen withdrawn from the other person's arm. This little excursion into vampire revelry was immediately effective; a negligible amount of blood was swallowed, we are told, as the subject became pale, yawned, said "I'm going" and lost consciousness—with, no doubt, a little warm blood dribbling from his lips. The scientific observation went on. The heart rate greatly slowed; no *QRS* complex was detected in the Lead II electrocardiogram for two consecutive periods of eleven and seven seconds; the forearm blood-flow was maintained throughout the period of the faint at a rate well above the resting level; the arterial pressure reading fell. After one and a half to two minutes consciousness was restored by raising the subject's legs; his ears and face were temporarily flushed, his heart action was irregular, with coupled beats, for several minutes, and he had nausea and retching with renewed slowing and irregularity of heart beats for about thirty seconds. Greenfield comments that the faint was mainly of emotional origin and that the changes observed in the circulation were similar to those in post-hæmorrhagic fainting. The combination of increased forearm blood-flow and reduced arterial pressure is clear evidence of vasodilatation of the peripheral vessels in the forearm; this, in association with skin pallor, almost certainly means that the vessels to the muscles were dilated. Dilatation of these vessels has been shown to depend on sympathetic vasodilator fibres; presumably, Greenfield states, the same is true of emotional fainting. Thus ends quite an interesting scientific observation. But two corollaries could be suggested. First, it would be interesting to have some comment, perhaps by a psychiatrist, on why blood and bloodletting cause an emotional response; is it fear of death or awe of life? Second, someone might send the story to Hollywood, and we could look forward to seeing, in glittering lights against the night sky, the magic words: "Physiological Fun or Dracula Rides Again."

VITAMIN D INTOXICATION.

THE dangers of massive or indiscriminate dosage of vitamin *D* or irradiated sterols have been pointed out at intervals during the last twenty years. The significance of pathological deposits of calcium in the tissues of infants who had received large doses of this vitamin was commented on in these pages a number of years ago. Hugh Chaplin, Lincoln D. Clark and Marian W. Ropes have published a review of 111 cases of vitamin *D* intoxication gathered from the literature, with the purpose of re-emphasizing the reality of this condition, especially as it is often not at first recognized.[1] These authors state that there is a wide variation in the susceptibility of individuals; this is no doubt true, though there must be a certain variation both in absorption and in utilization in different persons. The number of fatal cases is small in this series; five deaths were thought to be due wholly or in part to this factor. The syndrome as described by these writers conforms to the account given in the general literature, and includes general weakness and loss of weight, symptoms referable to the gastro-intestinal, nervous, renal and hæmatological systems, and other evidences of hypercalcæmia as proved by chemical, radiological and histological tests. Of course, it is not to be implied that all or even the majority of these signs will be present in a given patient. The production of toxic symptoms may also be accentuated by the ingestion of a diet containing high amounts of calcium, and by the taking of large doses of preparations containing vitamin *D*. Chaplin, Clark and Ropes point out that many such preparations are on the market which, either in liquid or capsule form, contain 50,000 units in each usual dose. They state that susceptible patients may show toxic signs after taking this dose for a period as brief as a few weeks. On the other hand more tolerant patients may take ten times this dose for six or twelve months before evincing symptoms. Seven case histories are given in this article to exemplify the observed signs and symptoms. The patients described are all of adult age, indeed, none was under thirty-six years, and several were old. They all suffered from some form of arthritis, though the chief presenting symptoms were not always specially characteristic of this. These case histories and the analysis of the others collected from the literature show a number of important points. Abdominal cramps may occur, with signs of gastric irritation and diarrhœa, variable mental symptoms may be observed, and evidence of renal pathological change is not uncommon. This last sign suggests one of the causes of susceptibility. Blood analysis may be expected to show the reciprocal changes in the content of calcium and phosphorus, with progressive nitrogen retention, and X-ray examination may demonstrate calcification in some of the soft tissues, with demineralization of the bones in advanced cases. It seems probable that milder examples of this syndrome might easily occur without detection if the high dosage of vitamin *D* was not pursued. One special sign observed by the authors in six out of seven cases was that of the so-called "band" keratitis, due not to the high dosage of the vitamin *D per se*, but to hypercalcæmia. In the same category are probably cysts containing calcium, which were also seen in some of their patients, usually in the periarticular regions, and near bursæ. This report shows that it may be easier to disturb the calcium balance of the body by well-meant treatment than is generally thought, and therefore it should be realized that highly potent preparations may be bought without prescription and taken over long periods with harmful results. Should the condition be recognized, the treatment consists of stopping the ingestion of vitamin *D*, keeping the calcium content of the diet low, avoiding over-exposure to sunlight and the administration of alkali and giving fluids freely. More important than these obvious measures is the maintenance of awareness that overdosage may occur.

[1] *The American Journal of the Medical Sciences*, April, 1951.

RESEARCH: SCIENCE OR ART?

IN a recent address to the International Congress of Clinical Pathology at London, Sir Lionel Whitby, discussing the science and art of medicine, held that the practice of medicine was both a science and an art.[1] He said that it could never become an exact science unless all variables could be estimated and allowed for, unless the personal element could be eliminated and unless each patient could be reduced to a standard form. Research is admittedly not completely comparable to the practice of medicine, but to understand the nature of research will be a help to the practitioner of medicine. One may ask whether research is a science or an art, and in seeking an answer we shall do well to read an important book recently published by W. I. B. Beveridge, late of Melbourne and now Professor of Animal Pathology at Cambridge. Beveridge leaves us in no doubt where he stands, for he entitles his book "The Art of Scientific Investigation"[2] and thereby throws out a challenge to the logician and the rationalist. "The great scientist", he declares, "must be regarded as a creative artist and it is quite false to think of the scientist as a man who merely follows rules of logic and experiment."

[1] *The Lancet*, July 28, 1951.

[2] "The Art of Scientific Investigation", by W. I. B. Beveridge; 1950. London, Toronto and Melbourne: William Heinemann (Medical Books), Limited. 8½" × 5½", pp. 190, with a few illustrations. Price: 10s. 6d.

The role of reason in research is not so much in the initial detection of new facts as in their verification, interpretation and development. Beveridge continues: "In formal education the student is implicitly, if not explicitly, led to believe that reason is the main, or even the only, means by which science advances. This view has been supported by the conventional conception of the so-called 'scientific method' and by some logicians who have little real understanding of scientific research. In this book I have tried to show the error of this outlook and have emphasised the limitations of reason as an instrument in making discoveries. I have not questioned the belief that reason is the best guide in known territory, though even here the hazards in its use are probably greater than generally realised. But in research we are continually groping beyond known territory and here it is not so much a question of abandoning reason as finding that we are unable to employ it because there is not sufficient information available on which to use it properly."

While reason is of little use in the making of discoveries, it finds a valuable place at a later stage, in the formulation of hypotheses, in an assessment of the correctness of ideas, in the planning of generalizations and finally in the finding of extensions and applications of a discovery. Its role is well illustrated by the story of penicillin. Between the work of Pasteur and Joubert in 1877 and that of Florey and his associates in 1939, there had been literally dozens of reports on antibacterial products produced by bacteria and fungi. Some had even been used therapeutically and good results claimed. But all these empirical discoveries were of little consequence until Florey, by a deliberately planned, systematic attack on the problem, produced penicillin in a relatively pure and stable form and so was able to demonstrate its great clinical value.

Beveridge regards the hypothesis as the principal intellectual instrument in research. Its function is to indicate new experiments and observations, and it therefore sometimes leads to discoveries even when not correct itself. In fact most hypotheses prove to be wrong, whatever their origin. As W. H. George graphically expresses it: "Even with men of genius, with whom the birth rate of hypotheses is very high, it only just manages to exceed the death rate." Beveridge devotes a chapter of his book to the importance of chance in research. He quotes many examples to illustrate this and goes on: "Probably the majority of discoveries in biology and medicine have been come upon unexpectedly, or at least had an element of chance in them, especially the most important and revolutionary ones. On reflection this is not surprising, for if a new fact is revolutionary it could scarcely have been foreseen from past knowledge." But more than chance is involved in discovery. "The rôle of chance is merely to provide the opportunity and the scientist has to recognise it and grasp it. . . . To interpret the clue and grasp its possible significance is the most difficult phase of all and requires the 'prepared mind'. . . . Anyone with an alertness of mind will encounter during the course of an investigation numerous interesting side issues that might be pursued. It is a physical impossibility to follow up all of these. The majority are not worth following, a few will reward investigation and the occasional one provides the opportunity of a life time. How to distinguish the promising clues is the very essence of the art of research." The travail that may attend the development of a chance observation into an accepted practice is illustrated by the story of Jenner. Thirty years elapsed after he was told that an attack of cowpox gave immunity to smallpox before he vaccinated a boy from a pustule on the hand of a milkmaid.

"Imagination is of great importance", writes Beveridge, "not only in leading us to new facts, but also in stimulating us to new efforts, for it enables us to see visions of their possible consequences. Facts and ideas are dead in themselves and it is the imagination that gives life to them. But dreams and speculations are idle fantasies unless reason turns them to useful purpose." In support of this opinion Beveridge appeals to Tyndall—"With accurate experiment and observation to work upon, imagination becomes the architect of physical theory"—to Planck—"This imaginative vision and faith in the ultimate success are indispensable. The pure rationalist has no place here"—and to Einstein—"There is no logical way to the discovery of these elemental laws. There is only the way of intuition, which is helped by a feeling for the order lying behind the appearance." Intuition is defined by Beveridge as "a clarifying idea that springs suddenly into the mind". It by no means always proves to be correct. Allied to it is scientific taste. Anyone who has this simply feels in his mind that a particular line of work is of interest for its own sake and worth following, perhaps without knowing why. How reliable his feelings are can be determined only by the results.

If research is an art depending largely on chance, on imagination, on intuition, to what extent can it be taught? On this Beveridge expresses himself thus: "The rare genius with a flair for research will not benefit from instruction in the method of research, but most would-be research workers are not geniuses, and some guidance as to how to go about research should help them to become productive earlier than they would if left to find these things out for themselves by the wasteful method of personal experience." Some things certainly can be taught—the value of the controlled experiment, the need for much thought in the planning of experiments, the importance of biometrics, meticulous care in note-taking.

The controversial topic of planning in research is handled clearly by Beveridge. There are three levels to be considered. The first is the actual conduct of an investigation by the worker engaged in the problem. This corresponds with tactics in warfare. It is short term and seldom goes far beyond the next experiment. The responsibility for tactical planning is best left to the individual scientist. But he should not be expected to adhere in detail to the programme of work that has been drawn up, for many discoveries have arisen from following a side track indicated by an unexpected observation. The second level involves planning further ahead on broad lines and corresponds with strategy in warfare. Planning at this level is not confined to the man engaged in the problem, but is often also the concern of the research director and the technical committee. Finally there is planning of policy. This type of planning is mostly done by a committee which decides what problems should be investigated and what projects or workers should receive support. Planning at the strategic and policy levels places a heavy responsibility on the planners, and is likely to be successful only when entrusted to people who have a real understanding of research as well as a good general knowledge in science. "One must not", Beveridge continues, "confuse the planning of research with the planning of individual experiments. No one would dispute the advisability of devoting great care to the planning of experiments and carrying them through according to plan." "Plans and projects are in order for tackling recognised problems, that is to say, for applied research, but science also needs the independent worker who pursues pure research without thought of practical results." The conclusion of the matter is that "research can be planned but discovery cannot".

A good deal of space has been given to a discussion of Beveridge's book because he pursues his study of the art of research with wit and wisdom. His analysis of the factors that underlie it is keen, his thinking is clear and sustained, his conclusions are sound and judiciously expressed. Senior students in science should be introduced to this book—it might well become for them a standard text—and its reading is urged on medical graduates who are engaged in, or associated with, research. As well as being very readable, it is also extraordinarily quotable. A wealth of anecdote illustrates each aspect of research. The great personalities of science have been laid under tribute to tell of their discoveries, their methods and their thoughts, and the author himself has fathered many an aphorism.

Abstracts from Medical Literature.

DERMATOLOGY.

Local Application of Œstrogens for Acne Vulgaris.

I. SHAPIRO (*Archives of Dermatology and Syphilology*, February, 1951) states that evidence that *acne vulgaris* is primarily an endocrine disturbance may be summed up as follows: (*a*) the usual incidence is at puberty; (*b*) the premenstrual flare-up of the lesions occurs when the œstrogen level is lowest; (*c*) production of acne occurs in females who are given androgen; (*d*) acne is not found among eunuchs; (*e*) acne is much commoner among males. These observations indicate that androgens produce pilo-sebaceous activity, and further work suggests an increase in the ratio of circulating androgens to œstrogens as a causative factor in *acne vulgaris*. A preliminary study indicated the value of cream containing œstrogens applied locally in the management of relapsing *acne vulgaris*. Ten men and fifteen women with the usual signs of *acne vulgaris* were studied. Their condition had proved refractory to the usual methods of treatment. Local applications were made of œstrogen in the form of a cream which contained 2·5 milligrammes of water-soluble conjugated œstrogenic substances (equine) expressed in terms of sodium œstrone sulphate per gramme of vanishing cream base. The average dose was five milligrammes of œstrogenic substances applied to the locally affected areas once daily or divided into morning and evening applications. Therapy was continued for from one to seven months. The total amounts of œstrogenic substances applied ranged from 150 to 700 milligrammes. The results are tabulated. In fifteen cases the results were good. Illustrative cases are given. No harmful effects were noted after four or five months' application of œstrogens.

Quinidine-Induced Exfoliative Dermatitis.

D. R. TAYLOR AND R. POTASHNICK (*The Journal of the American Medical Association*, March 3, 1951) report a case of exfoliative dermatitis resulting from the administration of quinidine. They refer to previously published reports of cases of anaphylactic shock and of thrombocytopenic purpura following quinidine therapy, as well as of a fixed eruption occurring in a man aged sixty-four years, in whom on a subsequent occasion a generalized scarlatiniform rash developed in addition. The authors' patient rapidly improved each time on withdrawal of quinidine, and the rash promptly recurred on readministration of the drug.

Macrocytic Anæmia and Impaired Liver Function in Dermatoses.

S. AYRES, JUNIOR, S. AYRES, THE THIRD, AND J. I. MIROVICH (*Archives of Dermatology and Syphilology*, December, 1950) state that Stryker and Halbeisen in 1945 called to the attention of dermatologists a relationship between certain dermatoses and nutritional disorders as revealed by macrocytic anæmia. It was pointed out that a routine blood examination, including red cell count, white cell count and hæmoglobin and colour index determinations, might fail completely to detect the presence of macrocytic anæmia of nutritional origin caused by a deficiency in one or more of the vitamin *B* complex factors. Only by measuring the mean corpuscular volume and the mean corpuscular hæmoglobin concentration could this important nutritional disturbance be detected. During the past two years the authors have encountered 27 cases of obscure inflammatory dermatoses in which the picture of macrocytic anæmia was found, sometimes suggested by the history, the general appearance of the patient or the presence of a smooth red tongue, amongst other indications. Both the skin lesions and the blood picture have shown gratifying responses to intragluteal injections of crude liver extract containing folic acid, together with high potency vitamin *B* complex and iron administered orally, and more recently to injections of vitamin B_{12}. Liver function tests were carried out in eight of the cases, and in each a mild to severe disturbance in liver function was revealed. Treatment consisted in the intramuscular injection of two millilitres of crude liver extract containing two milligrammes of folic acid per millilitre. In addition vitamin *B* complex derived from yeast and liver with added folic acid and iron was administered by mouth. Another series of 88 patients with various dermatoses, especially of an eczematous type, were found to have impaired liver function. In the majority of these cases cure or alleviation of the dermatoses followed the use of liver supporting therapy. A diet of low fat and high protein and carbohydrate content is recommended, with the exclusion of alcohol.

Para-Aminobenzoic Acid for Dermatitis Herpetiformis.

C. J. D. NARAFONETIS, E. B. JOHNWICK, L. KIRKMAN AND A. C. CURTIS (*Archives of Dermatology and Syphilology*, January, 1951) state that their reason for undertaking a trial of para-aminobenzoic acid therapy for *dermatitis herpetiformis* had its origin in the study of its effect in *lupus erythematosus*. *Lupus erythematosus* is often associated with hypersensitivity to sunlight and patients receiving sulphonamide drugs not infrequently manifest sunlight sensitivity. Since para-aminobenzoic acid and sulphonamides are metabolite antagonists, it was reasoned that para-aminobenzoic acid might have an opposite and beneficial effect in cases of *lupus erythematosus*. This idea was tried clinically with good results in cases of *lupus erythematosus*, so it was decided to try para-aminobenzoic acid in other conditions in which light hypersensitivity might be a factor. One of the authors had had a patient with *dermatitis herpetiformis*, the activity and discomfort of whose lesions were increased by exposure to sunlight. Since she no longer responded to the usual forms of therapy it was decided to administer a trial course of para-aminobenzoic acid. The gratifying response observed in this case led to the treatment of additional patients with *dermatitis herpetiformis*. Sixteen of these were treated and their case reports are given in detail. From these reports evidence is given that the administration of large doses of para-aminobenzoic acid caused partial to complete suppression of *dermatitis herpetiformis* in all 16 cases. The improvement required 18 to 24 grammes daily of either potassium or sodium para-aminobenzoate, although continued control was sometimes achieved with smaller doses. The drug caused suppression of the lesions and subsidence of the pruritus. It usually required two or three weeks of therapy to achieve this result. Relapse began to appear about ten days after the cessation of the treatment. Some patients gradually reduced the daily intake to 12 to 15 grammes per day with satisfactory suppression of the disease. This treatment was maintained as long as thirty months without any serious toxicity being encountered. Nausea, occasionally associated with vomiting, was the main untoward effect. Repeated leucocyte counts failed to reveal any cases of agranulocytosis, but the authors advise regular periodic checking of the blood. Spontaneous hypoglycæmia may arise. Patients should eat well to avoid this complication. If anorexia or nausea occurs, the drug should be discontinued till the patient is eating normally.

Lymphocytoma Cutis.

A. B. LOVEMAN AND M. T. FLIEGELMAN (*Archives of Dermatology and Syphilology*, February, 1951) state that in 1906 Jadassohn presented the first case of what was later to be *lymphocytoma cutis*, and in 1922 commented that his original patient was still living, that the skin lesions were unchanged and that no signs or symptoms of leuchæmia had developed. In 1921 Kauffmann-Wolf reported two cases in children and coined the name "lymphocytoma"; they did not consider that the condition was related to leuchæmia. Since then many cases have been reported. The authors' two cases of localized multiple type *lymphocytoma cutis* are reported. They discuss the differential diagnosis and histological characteristics and emphasize the necessity for the existence of a high index of suspicion before the differential diagnoses is considered. The lesions can resemble *epithelioma adenoides cysticum* and also rhinophyma. The diffuse swelling with localized subcutaneous mass felt on palpation brings to mind a sebaceous cyst. Involvement of the ear lobes is very common in *lymphocytoma cutis*. Any differential diagnosis of *lymphocytoma cutis* should include *lupus miliaris disseminatus*, the localized type of Spiegler-Fendt sarcoid (Boek), keloid, malignant lymphoblastoma, *lymphangioma circumscriptum*, and particularly benign cystic epithelioma. An accurate diagnosis can be confirmed only by correlation of the clinical and histological observations along with repeatedly negative results from hæmatological and bone marrow studies. The necessity for observation for many years to rule out the malignant lymphoblastomata is stressed. The clinical and histological similarity of certain localized types of Spiegler-Fendt sarcoid and *lymphocytoma cutis* is emphasized. The authors state that the lesions of *lymphocytoma cutis* are extremely radiosensitive; in one case, the administration of 150 volts (unfiltered), repeated three times at intervals of three weeks, brought about complete resolution of the

lesions. In the cases reported, regardless of the type of therapy employed, local recurrences in scar tissue and new lesions were frequently encountered.

UROLOGY.

Surgery for Conservation of Renal Parenchyma.

ELMER HESS *et alii* (*The Journal of Urology*, August, 1950) report that in the period from 1940 to 1948 inclusive they carried out 42 operations for conservation of renal parenchyma. Nineteen surgical repairs were carried out at the uretero-pelvic junction, four of which involved severance of large vessels without removal of the corresponding parenchyma. Thirteen partial resections of the kidney were carried out for localized calculi in otherwise anatomically normal kidneys. Three heminephrectomies were performed for stones in congenitally anomalous kidneys. Five upper poles were removed because of infection. Two heminephrectomies were performed in horseshoe kidneys, both for infection in the lower pole of one segment. In four additional cases of horseshoe kidney, the isthmus was divided, and attempts made to rotate the freed segments, with replacement of the ureter, so that it was rendered free from obstruction. In one of these cases the lower pole of one segment was still obstructing the ureter, and had to be resected to allow free play to the duct. There was no mortality in this series. The results were all satisfactory. The authors state that as regards technique, with particular reference to partial resection of the organ, to some extent technical improvisation must be relied on, but certain principles should be respected. The first is preservation of the blood supply. The authors dislike the application of temporary clamps to the pedicle; the fingers of the assistant are less traumatic, and are adequate. The second principle is preservation of parenchyma; deep mattress sutures pulling renal substance together to control hæmorrhage destroy much tissue. Capsular sutures should be sufficient, if the capsule can be split, and especially if a pad of fat or muscle is placed between the raw surfaces. In some cases of this type, postoperative excretion urograms suggest that the technique has allowed preservation of a maximum of functioning parenchyma.

True Priapism.

B. S. ABESHOUSE AND L. H. TANKIN (*The Urologic and Cutaneous Review*, August, 1950) consider that true priapism is a rare phenomenon. From the literature they have collected only 378 cases, which include four of their own. They state that the three main varieties are nervous, mechanical and systemic. The pathogenesis is dependent on the development of abnormal coagulative changes in the venous spaces of the *corpora cavernosa*, less often in those of the *corpus spongiosum*. Mostly, the material found in the corpora is thick, dark and grumous, and suggestive of pseudothrombosis. In a small proportion of cases, small clots, indicative of true thrombosis, were found. Priapism is usually encountered between the ages of twenty and fifty years. In the youngest group there is usually an associated blood dyscrasia, and in the oldest, a primary or secondary malignant infiltration of the *corpora cavernosa*. The chief signs and symptome are a firm, tense consistency of the corpora, with bluish discoloration, a unique position at an acute angle to the pubis, pain, urinary difficulty, and absence of sexual desire. The correct diagnosis is usually established by careful history and thorough examination, including complete blood count, investigation of blood chemistry and serological tests. True priapism seldom responds to conservative measures, except in cases of nervous origin, for example, reflex stimulation from disease of the lower part of the genito-urinary tract. Priapism of mechanical or systemic origin requires surgical treatment, namely, aspiration or incision and drainage of the *corpora cavernosa*. Priapism of malignant origin may demand amputation of the penis for relief. To prevent recurrence, the following prophylactic measures may have to be considered: (*a*) irradiation of the spleen, long bones and penis in leuchæmic priapism; (*b*) blood transfusion; (*c*) anticoagulant therapy; (*d*) rectal diathermy to improve circulation in the corpora; (*e*) stilbœstrol therapy.

Sterilization of the Infected Neurogenic Bladder.

IRVING SIMONS (*The Journal of Urology*, October, 1950) has undertaken an investigation to determine a good method for eradication of urinary tract infection in neurological patients, chiefly those with neurogenic bladders. He states that in such cases it is now recognized that bladder lavage is of doubtful value. This includes the use of tidal drainage, which is merely intermittent vesical lavage with the disadvantage that it requires a retention catheter. In any case such lavage is often doomed to failure since the upper part of the urinary tract is already infected. Since those infections are of long standing the ideal drug is one that can be given by mouth, one that is not irritating to the gastrointestinal or urinary tract, one that produces few, if any, untoward symptoms, and finally one which has a wide antibacterial range and to which resistance is not easily developed. In this study "Mandelamine" (methenamine mandelate) was used, since previous reports on its use in longstanding and protracted cases sounded encouraging. Of 174 patients with chronic neurological conditions studied, 122 were infected (70%). A single organism was found in 73% of cases, double infection in 27%. The organisms most commonly found were *Bacterium coli*, *Staphylococcus albus* and *Bacillus proteus*. "Mandelamine" was administered by mouth in doses of three to four grammes daily in divided doses; each tablet weighs 0·25 gramme. It tends to acidify the urine itself, but if the pH is above 7·0 one should use 2·0 grammes of acid sodium phosphate daily, until the pH is 6·0 or less. No local or general untoward symptoms were noted in this series in periods of administration up to four months, or even more. After much experience the authors prefer the large (four grammes) daily dosage. "Mandelamine" is not chemotherapeutically selective; it is effective against *Bacterium coli*, *Staphylococcus albus*, *Bacillus proteus* and practically all other organisms met in this series. Of the 122 cases of infection in this series, complete cure (sterilization) was obtained in 36 and duration of treatment was from one to two months. In this series failure occurred in 32 cases. It is important to note that the bacteria in these cases were of the same types as in the completely successful group. After study of the failure group the author concludes that the four grammes daily dose would be better right from the beginning.

Hyperparathyreoidism and Urolithiasis.

D. E. BEARD AND W. E. GOODYEAR (*The Journal of Urology*, November, 1950) state that the belief of the majority of urologists that hyperparathyreoidism is rare, and is an insignificant cause of renal calculi, is erroneous. During the last few years work by Albright and others has changed the criteria of diagnosis in this disease. Bone changes of *osteitis fibrosa cystica*, once regarded as necessary for diagnosis, are now known to be present in less than one-third of the cases. Further, the classical concept of multiple small bilateral renal calculi as a definite characteristic of the disease is no longer tenable. In 150 cases of urolithiasis studied by the authors, no less than 12 cases of hyperparathyreoidism were discovered, and this disease was proved to be the cause of the lithiasis. Greatly increased serum calcium levels were once considered the biochemical criteria of diagnosis, but now it is known that only a very slight elevation may occur in the disease, namely, 12 milligrammes per 100 millilitres, or even a little less. The symptoms of hyperfunction of the parathyreoid glands are vague, and often absent. Polyuria occurs in many cases; aching in the extremities and joint pain are common, and are often associated with weakness, fatigue and loss of appetite. So far as the gland is concerned, it is unusual for the adenoma to be palpable, since the tumour is quite small, and of the same consistency as the thyreoid gland. Inability to palpate a tumour in the patient's neck should not deter the surgeon from exploration when the chemical findings indicate the presence of the disease. Serum calcium levels are always raised, if only a little, if repeated reexaminations are made; the normal serum calcium level is 10 milligrammes per 100 millilitres with a range of 0·5 milligramme on each side. Serum phosphorus levels are lowered, but not always; the normal level is 3·5 milligrammes per millilitre with a range of 0·5 milligramme on each side. In addition to hypercalcæmia and hypophosphatæmia there occurs a constant hypercalcinuria. An output of 130 to 200 milligrammes of calcium in twenty-four hours is suggestive of the disease. The test is made on ambulatory patients only, as recumbency gives a "false positive" result. The calcium intake must be controlled for forty-eight hours prior to collection of the twenty-four-hour specimen, and should be maintained at 120 to 130 milligrammes daily. The authors describe fully their 12 cases of hyperparathyreoidism, found in 150 patients with urolithiasis. All the cases were proved by operation. The removed tumours weighed between one and four grammes with an average of about two grammes.

British Medical Association News.

SCIENTIFIC.

A MEETING of the Oto-Laryngological Society of Australia (British Medical Association) was held on June 4, 1951, in Sydney.

Pre-Bronchiectasis.

DR. H. B. HARWOOD (Sydney) read a paper entitled "Pre-Bronchiectasis" (see page 258).

DR. J. H. SHAW (Melbourne) said that Dr. Harwood's paper was very interesting in the facts it disclosed. It was difficult to say much about it from the average ear, nose and throat surgeon's point of view because the latter did not see the patients concerned in the early stage of the disease. It was not the general practitioner who needed it so much as the children's physician. The significance of early signs was not appreciated either by practitioners or by children's physicians, and ear, nose and throat surgeons were not likely to encounter a great many of the patients affected unless they were directed to a bronchoscopic clinic. It was very interesting to see the percentage of cases in which a pronounced improvement occurred. Although healing at the pre-bronchiectatic stage, there were in some of the more advanced cases changes which one would have thought irreversible. In later stages children were still capable of experiencing a great deal of amelioration. Dr. Shaw went on to say that the use of local anæsthesia in small children had certain disadvantages; he had always felt a certain degree of anxiety in the use of local anæsthesia in young children. It required the utmost care, as signs of toxicity were very slight and must be discerned immediately so that no further anæsthetic was administered. He would like Dr. Harwood's opinion, too, about the reaction to local anæsthesia shown by the patients under discussion. Dr. Harwood had not mentioned encountering any toxic effects.

DR. H. H. R. NASH (Perth) said that such a large proportion of the patients of the type under discussion had a definite allergic background that he would like to know whether anything was done about that allergy, and if so, in what way the problem was tackled.

DR. L. W. JOHNSTON (Melbourne) asked Dr. Howard how he dealt with those cases in which sinusitis was associated with bronchiectasis. Dr. Johnston had in the past tried to wash out the antra of children with a local anæsthetic, but he could not get near them, and he did not like the idea of a general anæsthetic.

DR. H. J. EIZENBERG (Sydney) said that he would like to ask Dr. Harwood whether he would consider performing a radical operation on the maxillary antra in those cases in which there was no response to intranasal antrostomy, as many patients still had discharging noses after intranasal antrostomy. In Dr. Eizenberg's opinion the bronchiectasis was not alleviated so long as the discharge continued. Pre-bronchiectatics had five monthly treatments, and bronchiectatics continued for fifteen months. What did Dr. Harwood use to wash out the bronchial tree? Did he use penicillin or Dr. Jackson's special solution, and did he favour the instillation of antibiotics through the bronchoscope?

DR. L. T. JOBBINS (Brisbane) asked Dr. Harwood what drugs he employed for premedication and local application.

DR. M. R. ROBERTSON (Lismore) asked what sort of sucker tube Dr. Harwood used.

DR. A. O. DAVY (Sydney) said that the question under discussion was very important for the teaching of students. It seemed that now, with the prominence given to bronchoscopic treatment and chemotherapy, the prevention of bronchiectasis might be almost attainable, and that should be emphasized, especially to students. It should be brought to their notice in a forcible way, as bronchiectasis had tended to become accepted as a thing which happened to some unfortunate children.

DR. A. B. K. WATKINS (Newcastle) said that radiographers should be trained in the condition under discussion. In Newcastle some years previously there had been reports on the condition, and gradually radiographers had been trained to mention a pre-bronchiectatic condition specifically in reports; so there was no difficulty in getting patients, as general physicians sent them along straight away. As soon as the doctor saw the word bronchiectasis in the written report, he soon saw to it that the child was directed into the proper medical channel.

DR. G. C. HALLIDAY (Sydney) said that he would like to ask whether the pre-bronchiectatic cases discussed developed into cases of fully fledged saccular type of bronchiectasis. He had been always taught that bronchiectasis was due mainly to peribronchial fibrosis. Did Dr. Harwood think that such a type could also develop from the so-called pre-bronchiectatic cases?

DR. E. P. BLASHKI (Sydney) said that the paper presented was definitely a milestone, because he remembered a meeting of the British Medical Association in Sydney at which the late Dr. R. Graham Brown had laid out clearly that sinusitis and bronchiectasis developed in that order. Bronchiectasis might be prevented at an early enough stage. A good deal of the success depended on the skill with which treatment was applied. Too often it was not carried out skilfully enough. The main thing found at bronchoscopy was the trauma done by a previous surgeon. Some of Dr. Harwood's success depended on his own personal skill. There was one point in the particular record presented to which Dr. Blashki would like an answer. It related to the pre-bronchiectatic conditions associated with collapse of small portions of the lungs. How wise was the constant use of atropine? It seemed to be doing the very thing to produce the condition which naturally occurred in the cases put before them by Dr. Harwood; so one wondered why the condition did not occur in the post-operative cases.

DR. G. C. SCANTLEBURY (Melbourne) said that before calling on Dr. Harwood to reply he would like to thank him very much indeed. He had enjoyed his paper very much. He did not need to read between the lines to realize what excellent work Dr. Harwood must do, and it was very good to hear Dr. Harwood's colleagues praising his thoroughness and good work. The nature of his work must be responsible for his very good results. The question of exactly how he achieved his results was Dr. Scantlebury's only criticism—the question of anæsthesia and solutions, and the amount of pressure which other people had referred to. Dr. Scantlebury noticed that Dr. Harwood seemed to clear up sinusitis much more easily than was Dr. Scantlebury's fortune. Maybe in the Sydney climate sinusitis was not so bad, but Dr. Scantlebury felt that in his experience with a series of cases such as had been described he would have found a greater amount of sinusitis and more difficulty in clearing it up. However, he thanked Dr. Harwood very much for his paper.

Dr. Harwood, in reply, referred first of all to the question of allergy. He said that some of the children affected had had skin tests performed, but not as a routine. A few of them had had injections of various things before coming up for treatment, and that apparently sometimes had a very good effect. One or two still showed signs of allergy, with watery discharge from the nose *et cetera*. The allergy always appeared to be a thing of which they would show some manifestations all their life. On the question of sinusitis, he said that if one wanted to wash out the sinuses of children round about seven years of age, in suitable cases that was carried out under local anæsthesia, but he would not hesitate to give a general anæsthetic to any bronchiectatics. The bronchi could be washed out at the end of the operation. Dr. Harwood said that radical antrostomy was not necessary for many of the children affected. Those with established bronchiectasis certainly should have it, and he quite agreed that unless that was done results would not be obtained with established bronchiectasis.

Dr. Harwood went on to say that ordinary methods of treatment were used. The patients were given premedication of heroin and atropine, but he was inclined to agree with Dr. Blashki about the use of the latter. A spray of "Decicain" and 1:1000 adrenaline solution was used. With very young children it should be just sprayed on the back of the throat. If they cried it was very much easier to examine them with the bronchoscope than when they were stubbornly silent and refused to open their mouths. However, most were quite cooperative. The solution used was Bledsoe-Fischer's solution. Dr. Harwood said that he did not wash out with penicillin. It seemed too pointless to wash out with penicillin once a month. The patients should be given penicillin in the lung every day, with bronchoscopy every week. That was changed if the organisms became insensitive to penicillin, and other antibiotics were used. Dr. Harwood could not see that penicillin inhalations and insufflations served any useful purpose. The inhalation went into the more healthy part of the lung. The solution could be emptied into the lung base, and the inhalations could not.

Referring to anæsthesia, Dr. Harwood said that he had carried out the procedure in just over 10,000 cases under local anæsthesia and had never had any trouble whatsoever until the last week when he had encountered his first difficulty.

Dr. Halliday had asked whether the condition described would progress to the more advanced stages. Dr. Harwood thought that there was quite a possibility that it would. In the pathology of today they realized that the old histology had to be revised a little, and the gloomy publications in some of the old books had to be brought more up to date.

Dr. Harwood said that the patients underwent bronchoscopy lying down and usually were running about a very short time afterwards. He liked them to stay in hospital for a few hours afterwards, but found it difficult to keep them there. He used ordinary hospital suction. He did not use suction on the bronchoscope. He put the solution down through the little canal at the side and that ran down the bronchoscope; he aspirated with the ordinary long sucker. He had some that went into the upper lobe bronchus. Granulations were painted with silver nitrate.

Dr. Blashki, in reply to Dr. Scantlebury, said that his objection to atropine was that it seemed that one of the most potent causes of bronchiectasis was the blocking of small tubes causing local collapse in a lobulus, which might become multiplied. If the secretion was thickened, and the giving of atropine would probably produce that, one set up the condition which might lead to obstruction and collapse. Except for the convenience of anæsthetists, there was not very much reason for atropine.

Dr. Harwood referred to Dr. Blashki's suggestion that secretion might be thickened, but commented that in the procedure under discussion a solution was used for washing out that loosened the secretion.

SIR CHARLES HASTINGS CLINICAL PRIZE ESSAY COMPETITION.

THE Sir Charles Hastings Clinical Prize Essay Competition is established by the British Medical Association for the promotion of systematic observation, research and record in general practice. The competition has been extended by the addition of a second prize known as the Charles Oliver Hawthorne Clinical Prize. The following are the regulations governing the awards:

1. The Sir Charles Hastings Clinical Prize, consisting of a certificate and 50 guineas, will be awarded for the best essay submitted.

2. The Charles Oliver Hawthorne Clinical Prize, consisting of a certificate and a sum of money slightly less than the amount of the Sir Charles Hastings Clinical Prize, will be awarded for the second best essay submitted.

3. Any member of the association who is engaged in general practice is eligible to compete for these prizes.

4. The work submitted must include personal observations and experiences collected by the candidate in general practice, and a high order of excellence will be required. If no essay entered is of sufficient merit no award will be made. Candidates in their entries should confine their attention to their own observations in practice rather than to comments on previously published work on the subject, though reference to current literature should not be omitted when it bears directly on their results, their interpretations, and their conclusions. It is suggested that essays should consist of from 3000 to 10,000 words.

5. Essays, or whatever form the candidate desires his work to take, must be sent to the Secretary, British Medical Association, British Medical Association House, Tavistock Square, London, W.C.1, not later than December 31, 1951.

6. A study or essay that has been published in the medical Press or elsewhere will not be considered eligible for a prize, and a contribution offered in one year cannot be accepted in any subsequent year unless it includes evidence of further work. A prizewinner in any year is not eligible for an award of either of the prizes in any subsequent year.

7. If any question arises in reference to the eligibility of the candidate or the admissibility of his or her essay the decision of the Council on any such point shall be final.

8. Preliminary notice of entry for this competition is required, on a form of application to be obtained from the Secretary.

9. Each essay must be typewritten or printed and must be accompanied by a sealed envelope, enclosing the candidate's name and address, firmly affixed to the essay.

10. The writer of an essay to whom a prize is awarded may, on the initiative of the Science Committee, be requested to prepare a paper on the subject for publication in the *British Medical Journal* or for presentation to the appropriate section of the annual meeting of the association.

11. Inquiries relative to the prize should be addressed to the Secretary.

Public Health.

VENEREAL DISEASES IN NEW SOUTH WALES.

THE following figures supplied by the Department of Public Health, New South Wales, show the total number, with an analysis, of notifications received of venereal disease infections in New South Wales for each of the past five years.

Analysis of Venereal Disease Notifications in New South Wales.

Data.	1946.	1947.	1948.	1949.	1950.
Total venereal disease notifications	5401	5011	4199	3043	2453
Total males affected	4572	4324	3580	2625	2074
Total females affected	829	687	619	418	379
Total cases of syphilis	1024	1010	1107	1033	627
Syphilis, primary and secondary	532	518	618	507	342
Syphilis, other	492	492	489	526	285
Cases of gonorrhœa	4163	3790	2884	1836	1657
Cases of other venereal disease	214	211	208	174	169
Rate per 100,000 of population of gonorrhœa	142·3	127	95·1	58·9	51·1
Rate per 100,000 of population of syphilis, all types	35·0	33·8	36·5	33·2	19·3
Rate per 100,000 of population of syphilis, primary and secondary	18·1	17·3	20·4	16·3	10·5

While it is realized that a number of infections are probably not notified, the figures supplied give an indication of the decline taking place in the incidence of these diseases, particularly gonorrhœa.

The use of penicillin in the treatment of gonorrhœa has had the effect of greatly reducing the period of infectivity of the carrier of disease, and, as a result, less infection is spread. Another factor of importance is that there is less chronic infection in females, and so a smaller reservoir for gonococcal infection exists.

Total Numbers of Notifications of Venereal Disease in New South Wales.

Disease.	1946.	1947.	1948.	1949.	1950.	1950.				1951.	
						January-March.	April-June.	July-September.	October-December.	January-March.	April-June.
Gonorrhœa	4163	3790	2884	1836	1657	526	336	349	446	335	285
Syphilis (total)	1024	1010	1107	1033	627	185	135	171	136	108	122
Syphilis, primary and secondary (in total)	532	518	618	507	342	112	73	90	67	51	53
Other venereal diseases	214	211	208	174	169	40	41	47	41	46	42

Out of the Past.

In this column will be published from time to time extracts, taken from medical journals, newspapers, official and historical records, diaries and so on, dealing with events connected with the early medical history of Australia.

PAINLESS OPERATIONS.[1]

[*Australian Medical Journal*, June 1, 1847.]

The human mind has a continual propensity to hunt after and adopt novelties, and to exaggerate their importance, too frequently in an inverse ratio to their real value. To this charge the members of our profession are not seldom, it must be confessed, obnoxious. The pretensions of mesmerism to the power of rendering patients insensible to pain during the performance of those operations which the imperfections of our art still render necessary to preserve life or to make it endurable, never received general sanction amongst us: but the idea once excited appears to have taken, for the time at least, a strong hold upon the minds of some of our brethren. There is a fashion in medicine, as in other, less important matters, and the rage just now seems to be after the means of rendering the use of the knife and other formidable instruments in the armamentum chirurgicum, free from pain and therefore and thereby, less dreadful and dreaded by those whose fate it is to be obliged to submit to their "sharp medicine". While we applaud a rational search after such means, if such exist, we would warn our professional confrères from the too hasty adoption of any measures for this purpose which may be offered for their acceptance. In former times it was customary to stupefy with narcotics a patient about to be operated upon, but for a long time past the practice has been abandoned. The plan now proposed and which has been imported into Great Britain from America is to intoxicate the patient by causing him to inhale the vapour of sulphuric ether. Now, although the inebriation produced in this way is very evanescent, it is still inebriation, and it does not appear to us of much consequence, if the operatee must be made drunk, what the intoxicating medium may be. For the minor operations in surgery we should consider any such measure unnecessary—in the more important ones we think it will be found not infrequently conducive to secondary consequences of a grave character, even if it did not, in many constitutions, produce immediate effects of a mischievous nature. Few practitioners, we imagine, would willingly, if they could avoid it, operate on a patient dead drunk from liquor, yet we here find it seriously proposed and practised, wilfully to make him drunk previous to the operation!

We have no hesitation in predicting for this process a transient popularity: it will have its day ultimately to be abandoned as useless or injurious. Meantime, some of the cases in which the ether has been used afford a curious illustration and confirmation of Dr. Marshall Hall's theory of the reflex action of the spinal nerves, for during the extraction of teeth, although the brain was unconscious of pain, "the features assumed an expression of pain and the hand was raised".

[1] By courtesy of the Mitchell Library, Sydney.

Correspondence.

THE ACADEMIC ASPECT OF MEDICAL EDUCATION.

SIR: I read with considerable interest Dr. Neville G. Sutton's thoughtful comments on my paper on this subject. His criticisms appear to reduce themselves to two.

The first concerns my contention that it is not necessary for a teacher to be a good practitioner. Let me repeat that the hospitals are full of excellent practitioners from whom the students can gain all the practical proficiency they need. It is not technique but ideas that the world requires, and in the choice of the head of a department the man of ideas must stand ahead of the man of purely practical skill. Of course, the man of ideas frequently does possess practical ability as well, and that is all to the good. But the ideas must precede the techniques.

The second criticism is that in stating that medical schools exist to teach medical sciences, not medical practice, I am refusing to face realities. Realities, I take it, are things as they exist today. There is plenty of dissatisfaction with medical education as it exists today, and I feel no need to apologize for presenting a case for trying to make it better for tomorrow. In the time available in the clinical years we cannot expect to produce anything more than people soundly grounded in medical principles, in the hope that with practical experience later they will become sound practitioners. The facts that most countries require an extra university examination of proficiency to practise, and that New South Wales, Queensland and Great Britain will be requiring a year's residence in hospital after graduation, are all evidence of the fact that our present attempt to turn out proficient practitioners straight from the medical school is unsatisfactory. In other words, the present compromise between medical science and medical practice has failed. This is one of the realities that Dr. Sutton apparently refuses to face.

Under the circumstances I shall continue to maintain that the job of the medical schools is to teach medical sciences. Those properly trained in the sciences will have no difficulty in acquiring the practical ability afterwards, and they will be the better practitioners for the extra science.

Perhaps what I have advocated is a university ideal, but surely it is better to have some such ideal in mind when planning a medical course than merely to muddle along patching up our present course with purely *ad hoc*, and usually badly conceived, measures as the necessity arises.

Yours, etc.,

The University of Adelaide, A. A. ABBIE.
Adelaide,
July 30, 1951.

A MORE REALISTIC VIEW OF TUBERCULOSIS.

SIR: In regard to Dr. Short's letter published in the journal (July 28, 1951), may I make the following comments?

I am sure we all share Dr. Short's disapproval of a "travelling clinic [wandering] through the State, and like a travelling circus pitch camp where the 'takings' are likely to be worth while, and then, passing on, leave a few worried people to be further investigated".

Unfortunately, Dr. Short's criticism contains the implication that this is in fact what happens. Since the only "travelling clinic to wander through the State" belongs to our association, may I inform Dr. Short that one of our doctors permanently accompanies the mobile clinic in its travels and conducts as many investigations as possible, on the spot? In addition the patients' sputums are sent to our clinic in the city, and when necessary, the patients themselves are encouraged to attend this clinic for gastric lavages *et cetera*.

Their worries are not different from the worries of other patients found to have tuberculosis in the cities, through clinics or hospitals, or through examination by private practitioners. The worry of having tuberculosis or even of being a tuberculosis suspect is a reaction common to all people involved, and it would be interesting to hear of methods whereby such a natural reaction could be eradicated.

We do not "pitch camp" where the "takings are likely to be worth while", but follow a systematic plan covering all country areas, irrespective of the potential "takings". I deplore, with Dr. Short, the necessity for such "takings", but a voluntary organization whose only income derives from charity could not exist without charging a nominal fee, whenever this can be afforded.

Our policy is to omit children from mass surveys except when, despite persuasion to the contrary, the parents insist on having their children X rayed.

Mass radiography should not be judged by the lesions which are missed, but by those which are detected. A patient whose lesion is missed is no worse off than a patient who had no X ray at all. Mass radiography offers the opportunity of a chest X ray to thousands of people who would otherwise never have had one. But for the travelling clinic in the country, many patients with advanced tuberculosis would continue, in their blissful ignorance, to infect their children and others in close contact with them.

I agree that there is room for a great deal of improvement in the antituberculosis campaign of New South Wales, but to say that the organizations dealing with this problem are

"dabbling" in tuberculosis is to deprecate a great effort through which already thousands of people find relief or cure and through which the mortality in this State shows an appreciable decrease.

Yours, etc.,
H. WILSON.

Anti-Tuberculosis Association of New South Wales,
Chest Clinic,
169 Albion Street,
Sydney.
July 31, 1951.

SIR: As one who had criticized Dr. Short's original letter on this subject, I hasten to congratulate him on his truly realistic view in the journal of July 28, 1951 (page 134). I am glad to note that his objections to mass radiography are more concerned with methods than principles, and endorse fully what he has said.

I should like to add a plea that the majority of cases of tuberculosis would be found by taking X-ray photographs of patients attending hospitals or already admitted, together with those seen by private practitioners.

Furthermore, as Dr. Short indicated, a preliminary Mantoux test properly executed would for the present avoid the unnecessary expense of an X ray in a very big percentage of cases.

Yours, etc.,
G. BRUCE WHITE.

135 Macquarie Street,
Sydney,
August 3, 1951.

CIRRHOSIS OF THE LIVER IN EX-PRISONERS OF WAR (JAPAN).

SIR: I was interested to read Dr. E. G. McQueen's account of hepatic cirrhosis occurring in four ex-prisoners of war (Japan), the clinical state being recognized for the first time some five years after the cessation of hostilities.

Whilst recognizing the value and interest in the production of cirrhosis by dietary deficiency alone in the experimental animal, one has never been convinced that this has been shown to be the case in man.

The history of these reported cases, the biochemistry and the biopsy illustrations could all be accounted for by a hepatocellular virus disease. It would seem to be worth while to ascertain the incidence of hepatic cirrhosis in all ex-prisoners of war (Japan) and to compare it with the incidence in the general population in Australia.

Yours, etc.,
S. J. M. GOULSTON.

231 Macquarie Street,
Sydney,
August 3, 1951.

A CLINICAL DESCRIPTION OF A DISEASE RESEMBLING POLIOMYELITIS SEEN IN ADELAIDE, 1949–1951.

SIR: I read with particular interest Dr. Pellew's article in your issue of June 30, 1951.

Under my care, for two months, I have had a patient (female, aged thirty-nine years) whose manifestations are identical to the clinical picture described in the Adelaide epidemic: long prodromal stage (at least three months), then true onset by sudden severe pharyngitis (*Staphylococcus aureus*), excruciating pain in the cervical part of the spinal column, generalized hyperæsthesia, vomiting, insomnia, hyperacusis, dysuria, stiff neck, stiff back, muscle weakness *et cetera*.

I wish to emphasize two essential facts: (i) Prolonged suprarenal deficiency. Very low blood pressure (maximum 60 millimetres of mercury, minimum 40 millimetres). (ii) Laboratory findings (*Institut officiel G. Bourret de Nouméa*). Cerebro-spinal fluid absolutely normal, but blood culture revealed after several days a special Listeria, which gave typical conjunctivitis to a guinea-pig. *In vitro* the germs proved to be sensitive to antibiotics and sulphonamides. The patient, being intolerant to sulphonamides and aureomycin, received "Chloromycetin", 10 grammes, and a total of 5,000,000 units of penicillin over a period of ten days.

This treatment was commenced before and continued after blood culture. In addition suprarenal therapy (*"Percortène"*) with anodynes and analeptics. The patient is now a convalescent, progressing slowly with partial recurrences of signs at longer intervals.

It is rather difficult for me to write in English, but I would be pleased to supply further information or receive any commentary which could help future patients inflicted with the same troubles.

Yours, etc.,
ÉDOUARD TRUBERT
(*Président de l'Association médicale de la Nouvelle-Calédonie*).

96, avenue Foch,
Nouméa,
Nouvelle-Calédonie.
July 23, 1951.

Post-Graduate Work.

THE POST-GRADUATE COMMITTEE IN MEDICINE IN THE UNIVERSITY OF SYDNEY.

Subscription Course of Annual Lectures.

THE Post-Graduate Committee in Medicine in the University of Sydney has decided to alter its policy of making special charges for attendance at lectures by overseas visitors, by making an annual charge of £2 2s. to cover all such lectures during the year and to include other specially arranged activities.

All members of the course will receive notices from time to time informing them of the programme, and special demonstrations and lectures will also be arranged. The fee of £2 2s. will cover the course for each year, ending June 30. Fees are deductible for taxation.

Mr. Naunton Morgan.

Mr. Naunton Morgan, F.R.C.S., surgeon to Saint Bartholomew's and Saint Mark's Hospitals, London, is visiting Australia as the official lecturer of the Australian Post-Graduate Federation in Medicine for 1951 and will give a course of three lectures on "The Rectum and its Diseases". These lectures will be given in the Stawell Hall, 145 Macquarie Street, Sydney, at 8.15 p.m. on the following dates: Tuesday, September 18; Monday, September 24; Wednesday, September 26.

A lecture will also be given at Newcastle on "Common Rectal Diseases" on Wednesday, September 19. Further details of this will be announced shortly.

Course in Cardiology.

The first annual refresher course in cardiology will be held in the Hallstrom Institute of Cardiology, in conjunction with the Post-Graduate Committee in Medicine in the University of Sydney. Dr. Paul Wood will act as guest lecturer of the institute for a period of three weeks at the invitation of the board of directors of the Royal Prince Alfred Hospital.

The course, entitled "Modern Concepts of Circulatory Disease", will extend over three weeks, and will be held at 2.30 p.m. each day from September 24 to October 5. A limited number of vacancies is reserved for practitioners other than members of the resident and honorary medical staffs of the Royal Prince Alfred Hospital. Applications from all those wishing to attend the course should be made to the Post-Graduate Committee in Medicine, 131 Macquarie Street, Sydney. The registration fee will be £5 5s.

Course in Occupational Medicine.

A course in occupational medicine, primarily intended for general practitioners, will be held at the School of Public Health and Tropical Medicine at the University of Sydney from September 26 to November 21, 1951, for nine half-day Wednesday periods. Five of the periods will be lecture-discussions to be held at the School of Public Health and Tropical Medicine. The four other afternoons will be devoted to field visits to industries. Dr. G. C. Smith, lecturer in industrial health, of the School of Public Health, will be the supervisor of the course.

The fee for enrolment will be £1 1s., and those wishing to attend should enrol as soon as possible with the Post-Graduate Committee in Medicine, 131 Macquarie Street, Sydney.

Films.

The Post-Graduate Committee in Medicine in the University of Sydney announces that the following films have been added to its library on loan, on behalf of the Minister for Italy in Australia: "Pericardectomy", "Laryngectomy", "Corneal Graft", "Glaucoma". Applications for the loan of these films should be made to the Post-Graduate Committee in Medicine, 131 Macquarie Street, Sydney. Telephones: BU 5238, BW 7483.

NUFFIELD FOUNDATION DOMINION TRAVELLING FELLOWSHIPS.

THE Nuffield Foundation will continue in 1952 its scheme of offering a number of travelling fellowships to Australian graduates. The awards available will be three fellowships in medicine, two fellowships in the natural sciences, one fellowship in the humanities, and one fellowship in the social sciences.

The purpose of the fellowships is to enable Australian graduates of outstanding ability to gain experience and training in the United Kingdom in their chosen fields, and to make contact there with scholars working in those fields, with a view to the Fellows' equiping themselves to take up senior posts in research and teaching in Australia.

The fellowships are intended for men or women of first-rate intellectual and personal qualities, who have already shown unusual capacity to advance knowledge and teaching in one of the fields concerned. Candidates must be Australian nationals, normally between the ages of twenty-five and thirty-five years, and must be university graduates holding, preferably, a master's or doctor's degree, and having subsequently had a year or more of teaching or research experience on the staff of a university or comparable institution.

A fellowship will normally be tenable for one year, but in exceptional cases may be extended for a further period of a few months. The fellowship will provide for return travelling expenses of a Fellow between his home residence and the United Kingdom, and, if he is married, similar expenses for his wife; an adequate allowance will be made for the Fellow's living and travelling expenses in the United Kingdom and for his academic fees, books and other incidental expenses, as well as a personal allowance. The total value of an award, including all travelling expenses, varies with needs and family responsibilities of the holder, but will in no case be less than £900.

A Fellow will be expected to resume residence in Australia on the completion of the fellowship.

Except with the express permission of the trustees of the foundation, a Fellow may not hold any other award concurrently with the fellowship.

A Fellow will be required to carry out, at centres approved by the trustees of the foundation, a programme of research work and training, similarly approved. Other work, paid or unpaid, may not be undertaken without the permission of the trustees. During the tenure of the fellowship a Fellow will not normally be permitted to prepare specifically for, or to take, examinations for higher degrees or diplomas awarded by bodies in the United Kingdom.

A Fellow will be required to submit to the trustees, at the end of the fellowship, a report on his work during the fellowship.

Should the trustees at any time find that a Fellow neglects or has neglected the obligations of the appointment, they shall have power immediately to terminate the fellowship.

The fellowships will be awarded by the trustees of the foundation on the recommendation of its advisory committee in Australia.

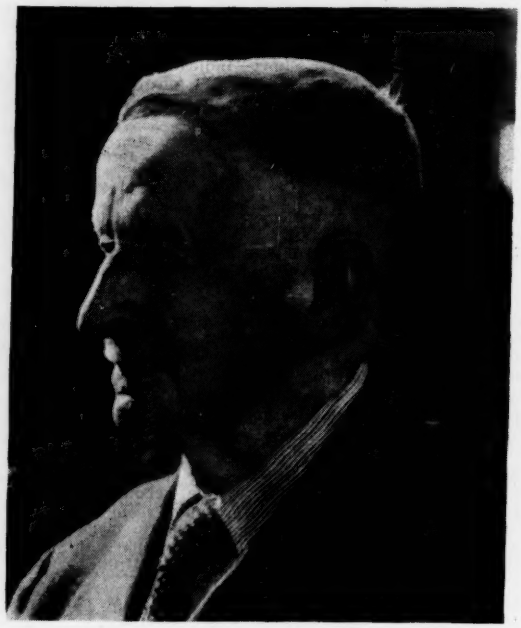
Applications for fellowships to begin in 1952 should be submitted not later than November 1, 1951, to the Secretary, Nuffield Foundation Australian Advisory Committee, c.o. The Australian National University, Canberra, A.C.T., from whom copies of the form of application may be obtained.

Obituary.

GEORGE WALTER BARBER.

WE are indebted to Dr. D. M. McWhae for the following account of the career of Dr. George Walter Barber.

Fifty-six years ago George Walter Barber came to Western Australia a young man of twenty-seven. For many years he practised on the goldfields which were still at the height of the boom following the discovery of gold. He there acquired a reputation as a sound and skilful general practitioner which he retained till the day of his death. Three years before the first World War he moved to Perth. Even in those early days he was interested in the Army Medical Service and he inspired a number of young medical men to join it.



In the first World War he commanded No. 2 Stationary Hospital which was stationed at Lemnos during the Gallipoli campaign and there handled a very large number of Australian casualties. Then towards the end of 1915 when the Australian Imperial Force was reorganizing in Egypt he was made Senior Medical Officer of Provisional Formations comprising the elements of two divisions. His organizing and administrative ability specially fitted him for this task, but it was in the field that his soldierly qualities and inspiring leadership reached their highest level.

He was Deputy Director of Medical Services of the 4th Australian Division from February, 1916, to April 8, 1918, during two years of intense fighting. His medical arrangements for the Hindenburg Line attack, the Battle of Messines and the Battle of Polygon Wood are fully described by Butler in the "Official History of the Australian Army Medical Services" and are models of careful organization and efficiency.

When in accordance with higher policy it was decided that an Australian officer should be Deputy Director of Medical Services of the Australian Corps, General Howse selected Barber; as Butler expressed it in his history "on account of a special aptitude for organization, and in temperament and administrative ability he was peculiarly fitted to take control of this office in the Corps". It was typical of Barber that he at once produced "standing orders" that can be studied today with profit by any Army Medical Corps officer. He took over his new duties at a critical time when the great German offensive to end the war was

about to be defeated, and he carried on until the end of the war. His medical arrangements for the Battle of Amiens and during the final advances to victory are also fully described by Butler.

Just before Barber left the 4th Division for the Corps, General McLagan, the commander of the division, told me that Barber's unfailing cheerfulness and optimism were a great asset, and that he and his officers were very sad at losing him; even in the dreadful conditions of the previous winter nothing could affect his cheerfulness, and Barber laughingly said that they were not nearly as bad as the goldfields in a dust storm, "and", said the General, "he really meant it".

His military service was of high distinction and he was awarded the C.B., C.M.G., D.S.O., V.D., and *Croix de guerre avec étoile*. He was mentioned seven times in dispatches. After the war Barber was Deputy Director of Medical Services in Western Australia until 1925 when he became Director-General of Medical Services with the rank of major-general. During the nine years in which he held this office he played the chief part in laying the sound foundation for the organization and training of the Army Medical Services which enabled them to function rapidly and efficiently in the last war. When war broke out he was appointed Chief Medical Officer of the Volunteer Defence Corps in Western Australia, and his comprehensive and detailed organization of the medical service of the corps could well be used as a pattern if it should ever become necessary to develop a similar service in future. Barber was a member of the Western Australian Branch of the British Medical Association and was president in 1921. He was vice-president of the Second Session of the Australasian Medical Congress (British Medical Association) at Dunedin in 1927, of the Third Session at Sydney in 1929, and of the Fourth Session at Hobart in 1934.

He spent the last seventeen years of his life in a quiet country practice in the hills overlooking the city of Perth, where he was regarded with affection and trust by his patients and by all who knew him.

He qualified in 1891 at the age of twenty-three years; he was not young when he left his wife and family to go to the first World War, and he worked as a doctor until within two days of his death at the age of eighty-three years, and this is a record of service that few can ever equal.

Dr. A. J. Collins writes: It was my privilege to serve under George Barber when he was A.D.M.S., 4th Division, and later as his D.A.D.M.S. when he became D.D.M.S., Australian Corps.

His great efficiency was apparent to me when I was a regimental medical officer and later an ambulance officer. He knew all his officers, visited them frequently and understood their problems and protected them from undue interference by combatant officers. He was a model A.D.M.S.

I worked more closely with him on the Corps Headquarters in 1918, from July onwards. This period coincided with the transition from trench warfare to the war of movement, which brought disaster to and final defeat of the German Army. The manner in which Colonel Barber adapted himself to his new charge was amazing. He quickly worked out a new system for field A.M.C. units; and as division leap-frogged through division, the evacuation of the wounded and the provision of medical supplies in the forward area were perfection. His great administrative capacity was recognized in all arms of the service, and was a model adopted by British corps in later offensives. I remember him as a man of great personal courage and uncanny skill and originality in directing all field medical operations.

Apart from all this he was a lovable gentleman of the old school. He was kindly and considerate of his juniors and extremely companionable. He was admired and liked by the combatant officers, and had the unswerving loyalty and affection of all who served under him.

GAVIN HOLME CAMERON.

WE regret to announce the death of Dr. Gavin Holme Cameron, which occurred on August 7, 1951, at Brisbane.

DISEASES NOTIFIED IN EACH STATE AND TERRITORY OF AUSTRALIA FOR THE WEEK ENDED JULY 28, 1951.[1]

Disease.	New South Wales.	Victoria.	Queensland.	South Australia.	Western Australia.	Tasmania.	Northern Territory.[2]	Australian Capital Territory.	Australia.
Ankylostomiasis	*	..	..	..	..	..	..	..	..
Anthrax	*	..	..	..	..	..	..	..	..
Beriberi	*	*	*	*	*	*	..	*	..
Bilharziasis	*	..	..	..	..	..	..	..	..
Cerebro-spinal Meningitis	3(1)	4(4)	1(1)	..	..	..	..	..	8
Cholera	..	..	..	..	..	..	..	..	..
Coastal Fever(*a*)	*	*	..	*	*	*	*	*	..
Dengue	*	*	*	*	*	*	..	*	..
Diarrhœa (Infantile)	..	..	7(7)	..	..	..	..	..	7
Diphtheria	5(2)	6(1)	8(6)	3(3)	3(2)	..	..	..	25
Dysentery (Amœbic)	*	..	..	..	..	..	..	..	..
Dysentery (Bacillary)	*	1(1)	1(1)	..	..	..	..	..	2
Encephalitis Lethargica	..	..	..	..	..	..	..	..	..
Erysipelas	*	*	*	1	*	*	..	..	1
Filariasis	*	..	..	..	..	..	..	..	..
Helminthiasis	*	..	*	*	*	*	*	*	..
Hydatid	*	1	*	*	..	*	*	..	1
Influenza	*	*	*	..	*	*	..	*	..
Lead Poisoning	*	*	*	*	*	*	*	*	..
Leprosy	..	..	1	..	..	..	..	..	1
Malaria(*b*)	*	..	..	..	..	..	..	..	..
Measles	*	*	*	43(21)	*	*	..	1	43
Plague	..	..	..	..	..	..	..	..	..
Poliomyelitis	16(8)	11(4)	22(14)	40(27)	3(3)	..	..	..	92
Psittacosis	*	..	*	..	*	*	*	*	..
Puerperal Fever	1(1)	..	..	2(2)	..	..	..	..	3
Rubella(*c*)	*	..	*	..	..	..	..	..	..
Scarlet Fever	17(12)	11(4)	6(4)	5(1)	10(6)	10(9)	..	..	59
Smallpox	..	..	..	..	..	..	..	..	..
Tetanus	*	..	..	*	*	*	*	*	..
Trachoma	*	..	*	*	*	*	*	*	..
Tuberculosis(*d*)	25(18)	29(25)	19(14)	13(7)	14(12)	2	..	..	102
Typhoid Fever(*e*)	1	..	..	..	..	..	..	..	1
Typhus (Endemic)(*f*)	..	..	3	..	2(2)	..	..	..	5
Undulant Fever	..	..	..	..	..	..	..	..	..
Weil's Disease(*g*)	*	*	..	*	*	*	*	*	..
Whooping Cough	*	*	*	2(1)	*	*	..	..	2
Yellow Fever	..	..	..	..	..	..	..	..	..

[1] The form of this table is taken from the *Official Year Book of the Commonwealth of Australia*, Number 37, 1946–1947. Figures in parentheses are those for the metropolitan area.
[2] Figures not available.
[3] Figures incomplete owing to absence of returns from the Northern Territory.
* Not notifiable.
(*a*) Includes Mossman and Sarina fevers. (*b*) Mainly relapses among servicemen infected overseas. (*c*) Notifiable disease in Queensland in females aged over fourteen years. (*d*) Includes all forms. (*e*) Includes enteric fever, paratyphoid fevers and other Salmonella infections. (*f*) Includes scrub, murine and tick typhus (*g*) Includes leptospiroses, Weil's and para-Weil's disease.

HARRY ROLAND HILL.

We regret to announce the death of Dr. Harry Roland Hill, which occurred on August 11, 1951, at Coonabarabran, New South Wales.

STANLEY GEORGE BRADFIELD.

We regret to announce the death of Dr. Stanley George Bradfield, which occurred on August 12, 1951, at Stanwell Park, New South Wales.

JOHN CADELL WINDEYER.

We regret to announce the death of Professor Emeritus John Cadell Windeyer, which occurred on August 15, 1951, at Wahroonga, New South Wales.

Naval, Military and Air Force.

APPOINTMENTS.

The undermentioned appointments, changes *et cetera* have been promulgated in the *Commonwealth of Australia Gazette*, Number 52, of July 19, 1951.

CITIZEN NAVAL FORCES OF THE COMMONWEALTH.

Royal Australian Naval Reserve.

District Naval Medical Officer.—Surgeon Commander James Estcourt Hughes is appointed District Naval Medical Officer, Port Adelaide, dated 21st September, 1950.

AUSTRALIAN MILITARY FORCES.

Reserve Citizen Military Forces.

Royal Australian Army Medical Corps.

The following officer is placed upon the Retired List with permission to retain his rank and wear the prescribed uniform:

5th Military District.—Captain A. T. H. Jolly, 7th July, 1950.

Australian Medical Board Proceedings.

TASMANIA.

The undermentioned have been registered, pursuant to the provisions of the *Medical Act*, 1918, as duly qualified medical practitioners:

Lennon, Evan Austin, M.B., B.S., 1949 (Univ. Queensland), Huonville.

Grant, Frank John, M.B., B.S., 1950 (Univ. Melbourne), Royal Hobart Hospital, Hobart.

Adey, William John, M.B., B.S., 1950 (Univ. Melbourne), Launceston General Hospital, Launceston.

Medical Appointments.

Dr. J. B. Cleland has been appointed to be an inspector of the School of Anatomy and to superintend the School established at the University of Adelaide.

Dr. N. D. Crosby has been appointed a member of the Central Board of Health of South Australia.

Dr. P. W. Verco has been appointed senior honorary assistant radiologist at the Royal Adelaide Hospital.

Dr. W. G. Norman has been appointed honorary assistant radiologist at the Royal Adelaide Hospital.

Dr. C. M. Gurner has been appointed honorary assistant radiologist at the Royal Adelaide Hospital.

Dr. K. B. Winter has been appointed honorary assistant to the director of the Psychiatric Clinic, Royal Adelaide Hospital.

Dr. C. H. Hembrow and Dr. L. T. Wedlick have been appointed members of the Masseurs Registration Board of Victoria.

Dr. B. Jarvis has been appointed a public vaccinator for the Shire of Kilmore, Victoria.

Dr. W. V. Rowe has been appointed a public vaccinator for the Shire of Kaniva, Victoria.

Dr. C. A. T. Edwards has been appointed a public vaccinator for the Shire of Numurkah, Victoria.

Dr. D. McL. Peace has been appointed a public vaccinator for the Shire of Hampden, Victoria.

Dr. R. M. G. Sobey has been appointed a public vaccinator for the Shire of Broadford, Victoria.

Diary for the Month.

AUG. 28.—New South Wales Branch, B.M.A.: Ethics Committee.
AUG. 30.—New South Wales Branch, B.M.A.: Branch Meeting.
SEPT. 4.—New South Wales Branch, B.M.A.: Organization and Science Committee.
SEPT. 5.—Victorian Branch, B.M.A.: Branch Meeting.
SEPT. 5.—Western Australian Branch, B.M.A.: Council Meeting.
SEPT. 6.—South Australian Branch, B.M.A.: Council Meeting.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Medical Secretary, 135 Macquarie Street, Sydney): All contract practice appointments in New South Wales.

Victorian Branch (Honorary Secretary, Medical Society Hall, East Melbourne): Associated Medical Services Limited; all Institutes or Medical Dispensaries; Australian Prudential Association, Proprietary, Limited; Federal Mutual Medical Benefit Society; Mutual National Provident Club; National Provident Association; Hospital or other appointments outside Victoria.

Queensland Branch (Honorary Secretary, B.M.A. House, 225 Wickham Terrace, Brisbane, B17): Brisbane Associated Friendly Societies' Medical Institute; Bundaberg Medical Institute. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 178 North Terrace, Adelaide): All Lodge appointments in South Australia; all Contract Practice appointments in South Australia.

Western Australian Branch (Honorary Secretary, 205 Saint George's Terrace, Perth): Norseman Hospital; all Contract Practice appointments in Western Australia. All government appointments with the exception of those of the Department of Public Health.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

All communications should be addressed to the Editor, THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales. (Telephones: MW 2651-2.)

Members and subscribers are requested to notify the Manager, THE MEDICAL JOURNAL OF AUSTRALIA, Seamer Street, Glebe, New South Wales, without delay, of any irregularity in the delivery of this journal. The management cannot accept any responsibility or recognize any claim arising out of non-receipt of journals unless such notification is received within one month.

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